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A MANUAL FOR

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BY

CHARLES LYMAN GREENE, M. D.

PROFESSOR OF THE THEORY AND PRACTICE OF MEDICINE IN THE UNIVERSITY OF MINNESOTA; ATTENDING PHYSICIAN, ST. LUKE'S HOSPITAL, THE CITY HOSPITAL AND THE ST. PAUL FREE DISPENSARY; MEMBER OF THE ASSOCIATION OF AMERICAN PHYSICIANS, THE AMERICAN MEDICAL ASSOCIATION, AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE; AUTHOR OF "THE EXAMINATION FOR LIFE INSURANCE AND ITS ASSOCIATED CLINICAL METHODS," ETC., ETC.

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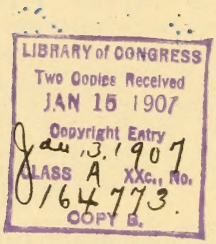

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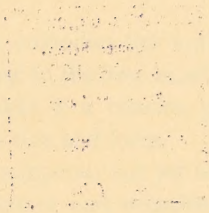

DEDICATION

TO MY STUDENTS, PAST AND PRESENT,

THIS LITTLE VOLUME IS DEDICATED.

PREFACE

This little volume embodies the author's conception of the type of book *most generally useful* to the over-taxed student and general practitioner and he has tried to make it a concise, practical and thoroughly modern handbook of convenient size and form.

It is not a mere compend, nor yet is it intended that it shall take the place of the many elaborate and valuable larger works relating to medical diagnosis, but it represents an effort to achieve the requisite completeness through direct statement, logical arrangement, and the avoidance of unproven or unessential theories and obsolete, time consuming or superfluous methods. To the same end, marginal notes and running page headlines supplement the full index and the illustrations, small in size though large in number, are believed to possess the virtues of simplicity and teaching value. The author hopes that he may have in some measure succeeded in meeting the exacting requirements of an undertaking, the difficulties of which are in inverse ratio to the size of the product.

For the kind suggestions of his confreres and especially to Drs. Haldor Sneve and Frank E. Burch for valuable services freely tendered in connection with the revision of manuscript sections dealing with the nervous system and the clinical examination of the eyes, he is most grateful. No less does he appreciate the courtesy and patience of his publishers and the technical resource which makes of 683 clearly printed and freely illustrated pages a volume which in shape, size, form and binding conforms to the specific purpose of the book.

CHARLES LYMAN GREENE.

SAINT PAUL, NOVEMBER, 1906.

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MEDICAL DIAGNOSIS.

PRELIMINARY REMARKS ON CASE-TAKING AND DIAGNOSIS.—*A diagnosis* means more than giving a name to a disease, and must include a correct estimate of the constitutional peculiarities of the patient, the nature and extent of pathologic changes, the effect of age, occupation, residence, habits, heredity, past ailments, and even the personal characteristics of the individual. Accurate diagnosis and intelligent prognosis are prerequisite to effective treatment.

Its scope.

From his text-books and the lecture room, the student learns the known types; at the bedside he soon realizes that variation from the type, and the personal equation must be carefully studied. *Diagnosis in medicine, even more than in surgery, must be based upon a sufficiency of facts, truthfully recorded, intelligently sifted, and viewed without bias or preconception.* Self-deception spells failure, as does that narrowed mental vision, which cannot see and appreciate new facts of later development, or the cowardice that fears to give up an erroneous preconception. Our knowledge of clinical symptoms in a given case depends either upon (a) *what we are told* or (b) *what we see or determine for ourselves*, i.e. they are either (a) *subjective* or (b) *objective*, the latter group including bacteriologic and chemic tests as well as physical signs. For the former, we depend upon the patient, and often encounter garrulity, stupidity, concealment, deceit or hypochondriacal exaggeration. If the patient is comatose or possesses no common language, we have no testimony save that of outsiders, and in any event must exercise sound judgment, keen discrimination and a certain facility in cross examination or we cannot give to these fallible yet valuable subjective data their proper weight.

Text book vs. Bedside.

The open mind.

Subjective vs. Objective.

"Hearsay evidence."

To make a case history full, accurate, yet concise, to elicit the salient facts and assign to each its proper value and perspective demands that the observer be full of knowledge, quick of perception and capable of avoiding both the shoals of omission and the rocks of verbosity. Certain routine inquiries should always be made, and the student should be extremely careful and painstaking in the days of his appren-

Tyro vs. Expert.

ticeship, though the skilled clinician can seize upon the cardinal points of his case and arrive at correct conclusions in a comparatively short time and with a minimum waste of words and ink. Certain single symptoms may name the disease, and such are termed *pathognomonic*, but woe to the man who is betrayed into the *habit* of making "snap" diagnosis. His opportunity passed when pathology came to her estate. *Diagnosis by exclusion* is a useful though roundabout and often unsatisfactory method of arriving at conclusions by a process of negation, the object being to find in the signs or symptoms presented by a given case, one or more inconsistent with the diagnostic symptom group of all diseases save one. Modern advance has greatly reduced the value of, and necessity for the method. Typhoid need no longer be laboriously established by negation, but in almost every case may be directly and promptly diagnosed by the Widal test and diazo-reaction. Its simulator, malaria, is no less positively known by the plasmodium as revealed by a blood examination. In some instances a *therapeutic diagnosis* is necessary, as for example, in dealing with an obscure or ancient suspected syphilitic infection when mercury and the iodides may banish all doubt. Many students and some practitioners habitually diagnose medical curiosities and such should remember that the doctrine of probabilities is against them, and thoroughly thresh out the variations of the prosaic and simple before assuming the long odds. On the other hand it falls to the lot of every man to encounter rarities and their recognition means added credit and reputation. The quack never hesitates to make a diagnosis, but the physician of parts, knowledge, and honesty must often make none or at best a provisional one, and wait for more light. The quack never acknowledges an error; the honest man whatever his ability must occasionally confess one. The more ignorant and dishonest the man, the more dogmatic and rapid his diagnoses, for with breadth and depth of knowledge comes its highest gift, a conception of its limitations. The physician must work methodically, deliberately, and with open mind, but once his opinion is formed, it should find emphatic expression, and every subsequent order and act be characterized by firmness and decision. If to these qualities he adds that modest self-confidence, born of fulness of knowledge and resource, his are the keys of the temple of fame. Such a man can and will deal with intelligent patients frankly and freely, he will not be afraid to call counsel, and need not feel that his after-conference with his consultant must be held in private according to a prevailing custom, more honored in the breach than in the observance. There are few

"Snap" diagnosis.

Direct vs. Indirect methods.

Therapeutic diagnosis.

Quack vs. Physician.

Consultation.

families of the intelligent sort in which the physician cannot find a confidant whom he can admit to his conferences, talk with freely and honestly, and thereby increase his usefulness and gain added support and a valuable co-operation. So also there are few physicians who will use a visit of courtesy as a means of self aggrandizement, or the humiliation of a fellow practitioner, and one who cannot deal fairly with all parties concerned is out of place in this day and generation. Doubts, fears and argument, however, are not for the sickroom, where a confident bearing and cheery countenance mean oftentimes more than a whole pharmacopœa.

Lay coadjutors.

Let the student beware of that method of study which leads him to arbitrarily commit to memory a long train of symptoms, without proper consideration of their general bearing, causes, or special and peculiar relations, rather let him learn to associate with each disease that which is peculiar and specific. A multitude of diseases, for example, are febrile but fevers are much alike as regards their general symptomology. This last he should learn thoroughly as of broad application, but stamp especially upon his memory the *peculiar type* of fever or definite variations that any given disease presents. Furthermore, every student should try to get a *clear mental photograph* of any ailment he is studying, and of the pathologic changes that underlie and explain its symptoms. In his mind's eye he should *see* the man with typhoid, the chart, the physiognomy, attitude, rose spots, and more than that the intestinal ulcers that underlie them. Such a method makes for thoroughness and for quickness of perception and inference and no man can take an intelligent, comprehensive and concise case history who has not mastered these principles.

The poll parrot method.

Mental photography.

Finally, and most emphatically, should it be stated that *case-taking, recording and reporting should be carried into every man's practice.* Old case books well kept are mines of knowledge and the science of medicine would be greatly enriched were the workers in city and hamlet alike to give to it reports of the unusual cases now for the most part allowed to pass without record.*

The value of the case book.

THE OUTWARD SIGNS OF DISEASE.

Every diagnosis commences with our introduction to the patient, and even the handshake may convey information of value. The physician

The first impression.

*For a fuller discussion of this topic the reader is referred to the delightful book of Byrom Bramwell entitled "Practical Medicine and Medical Diagnosis."

Go slowly.

should train his observation and would do well to emulate the example of his predecessors of a darker medical era, who were adepts by force of necessity. On the other hand, he should avoid hasty physiognomic diagnoses, for appearances are often misleading. Diagnosis in disease of the nose, throat, eye, ear and skin is essentially that of inspection, and throughout the whole range of medicine and surgery it plays an important part.

Diathesis.

FACIAL EXPRESSION.—Character and temperament, bad habits, diseases past and present, may stand revealed at a glance. Temperament is closely related to diathesis, but the latter implies a tendency to some special type of disease. The former has lost the prominent place it held in earlier times, and one need give little thought to its elaborate classification, though the "nervous," "bilious," "phlegmatic," "lymphatic," and "melancholic" types are often clearly defined and aid in diagnosis and prognosis alike. Who for example, has not encountered the "lymphatic" individual with a tuberculosis, which burns him to cinders, while the physician stands helpless to stay or cure? The

Malingerers.

expression helps us with malingerers, those forgers of symptoms who seek shelter under the hospital roof or wish to saddle damages for some spurious hurt upon the rich individual or corporation. In the hang

The "hang dog" air.

dog carriage and shifting eye of the youth, and his moist clammy hand we may read the story of sexual abuse. If melancholy has marked

Melancholia.

the patient for her own, he carries on his face the impress of her signet. Incipient insanity of other types may manifest itself in a peculiar expression of the eyes, "intensification of emotional expression," as it has been called, and a deceptively merry expression it often is.

Facies and speech.

General Paresis in its early stages often gives outward evidence. Lips, tongue, fingers and facial muscles are tremulous; the pupils are unequal and react to accommodation but not to light. The speech is slow, hesitant or explosive, consonantal words are difficult for the victim, and his delusions of grandeur are likely to find an outlet in his conversation. Everything of his is superlatively beautiful, simple or great, and he incomparable.

Facies.

Carcinoma of the Stomach is frequently associated with a curiously morose or saturnine expression well shown in the famous statue of Napoleon in his last days.* This, together with marked emaciation and a peculiar earthy color are most suggestive.

Congenital syphilis.

Syphilis.—The snuffling cry, weazened monkeyish face, fissured lips and raw nostrils and buttocks of a syphilitic babe are pathognomonic. The permanent upper central incisors of the older children are likely

*In the Corcoran Gallery, Washington.

to be peg-shaped, notched at their cutting edges, irregular and separated. Fine linear scars may radiate from the angles of the mouth. Keratitis and chronic otitis media often co-exist, and at any age a frog face (see illustration) may result from necrosis of the nasal arch. Syphilitic alopecia may be present, or have left its mark in a patchy grayness.

"Frog face."

Fig. 1.—Syphilis. Grey areas of past alopecia. Saddle-nose. Patient and his father both showed perforated palate.

Many cases of past syphilitic infection give no evidence that can be detected in the ordinary examination, the examiner being forced to rely upon the patient's statement in reply to a direct question which should be definitely and clearly put in the case of the men though manifestly impossible when dealing with women. One of the most easily recognized and pathognomonic syphilitic types is that combining alopecia or scattered areas of gray hair with saddle nose and perforated palate. A deeply fissured tongue *suggests* this disease and many cases of course present themselves with an active and unmistakable eruption or "the primary lesion."*

Syphilis in adults.

Perforated palate.

DRESS.—The quality of the clothing and its condition may suggest the position in life of its wearer as well as his occupation. Carelessly worn and disarranged garments, stained by food droppings may suggest mental change, particularly if noted in a person formerly neat and well and carefully dressed. An ammoniacal odor suggests incontinence or cystitis, and white stains upon the shoes may give a hint of an existing saccharine diabetes. In patients formerly stout who have recently lost greatly in weight the old clothes may be worn and be suggestively loose. On the other hand the development of an anasarca may make the patient appear to be bursting through his clothing. Edema of the legs is frequently suggested by loose lacing of the shoes, which may be cut and slashed for the same reason or the better to accommodate corns, bunions or gouty toes. In the various forms of pathologic gait the wearing away of the shoe in a particular part may be suggestive, this being particularly noticeable in the spastic conditions.

Suggesting brain trouble.

Urinary Disorders.

Recent weight loss.

Edema.

Paralysis.

THE COLOR OF THE SKIN.—The two conditions chiefly to be noted are pallor and cyanosis; the pale face may or may not mean true anæmia, and a *high color may co-exist with a low hemæglobin percentage*. More stress may be laid upon the color of the mucous membranes and

Sometimes misleading.

*See syphilis, pp. 475 to 481.

"Rosy chlorotics."

though certain degrees of pallor may exist there without anæmia even "rosy chlorotics" usually show a pale conjunctiva. Ordinarily, one may regard pallor of the cheeks, *lips*, tongue, throat, *ears*, *conjunctivæ* and finger nails as indicating a reduced hemoglobin percentage. The color varies markedly in different types of anæmia, being greenish yellow or almost waxy white in chlorosis or simple anæmia, a peculiar lemon yellow in pernicious anæmia, and a curious earthy tint in the profound secondary anæmia of malignant disease of the stomach.

Anæmic types.

Nephritis.

In Acute Bright's Disease the skin is white and an associated edema produces the peculiar and characteristic *pasty pallor*. *In parenchymatous nephritis* we meet with pallor or in its late stages a sallow or brownish hue. Most authors speak of the peculiar "fawn colored" skin as related to interstitial nephritis, but in the author's experience it is more often met with in cases which come under the "mixed" type of chronic kidney disease, or only in the late stages of the interstitial form, the earlier period being often associated with the ruddy or high colored ("clubman") countenance, and many of its victims appearing exuberantly healthy.

"Fawn color."

Cardiac disease.

Deceptive pallor.

The color in certain forms of heart disease is both interesting and important. *Aortic regurgitation* is usually associated with a pallor of the indoor worker type, there being rarely any marked true anæmia, but even in compensated regurgitant and obstructive disease of the mitral valve the color is often deceptively high, producing in girls especially what the laity regards as an exquisite complexion, though in older women and in men such redness of the cheeks is likely to be less generally distributed and more patchy. The skilled eye sees the duskiness of the underlying cyanosis which shows yet more plainly in the mucous membrane of the lips, the skin of the ears, nose and patella, and the nails whose pink is replaced by a darker hue or in extreme cases by a purplish or blackish gray.

False "color."

Cyanosis whether general or local, with or without true dyspnœa, ordinarily indicates obstructed venous return, deficient oxidation, or commonly both factors such as may result from ordinary suffocation, congenital heart disease, asthma, emphysema, pulmonary fibrosis, obstructed glottis, trachea or bronchi, as from foreign bodies, croup, laryngeal diphtheria, capillary bronchitis or broncho-pneumonia proper, mediastinal tumors, or other heart and lung lesions. It occurs also in acute diseases such as pneumonia, or pleurisy with either liquid or gaseous effusion, and to a slight degree, in severe acute bronchitis. Paralysis and spasm, particularly of the diaphragm, may produce marked

Underlying causes.

cyanosis as may the inhibition of efficient respiration by severe pain. No outward sign of disease exceeds cyanosis in importance and one short cut to diagnosis lies in the fact that *in its extreme form it exists in but two classes of walking patients*, viz.:— severe emphysema, in the adult, congenital heart disease, in the child. It is often associated with mere chilling of the body surface, hysteria, neuritis, etc. *Local vaso-motor relaxation* or paralysis is readily distinguished by the lack of turgidity of the venous trunks, and it should be remembered that certain forms of drug poisoning, especially acetanilide and its congeners nitro-benzol, etc., may account for an otherwise inexplicable and extreme cyanosis. Through excessive heat loss cyanotic areas are cold save in acutely inflamed areas. The slighter degrees are often unnoticed by the student but *pronounced cyanosis cannot be overlooked.* Of the many other departures from the normal tint one may mention the yellowish brown, dark brown or brownish black of *Addison's disease*, the peculiar bluish or blackish gray of *argyria* (chronic silver poisoning), the sallow tint of *malaria* and the varying yellows produced by *jaundice*. *Arsenical melanosis* may exactly simulate Addison's disease but usually diminishes under drug discontinuance. The yellowish brown patches of *pregnancy* (chloasma gravidarum) and pelvic disorders are common and easily recognized. Streaky pigmentation usually indicates scratching as the result of skin parasites or pruritic lesions. *Melano-sarcoma* is associated with a grayish or blackish skin and the urine shows melanin. A brownish or dirty gray spotted or patchy discoloration may occur in *hepatic cirrhosis* with or without jaundice and bronzing may be associated with combined cirrhosis and diabetes (bronze diabetes). Aside from its secondary color manifestations, *syphilis* in its tertiary and congenital form produces a peculiar sallow pallor not easily described, but easily learned by observation. So far as the author's observation goes it is met with chiefly in old cases of imperfectly treated syphilis, and is likely to be associated with other "reminders." Peculiar but indefinable forms of pallor may mark the *cocaine and opium* habitue, best observed in the opium dens or the special hospitals of our Pacific Coast towns or in the Orient. The author has observed cases in which the appearance was remarkably like that of chronic nephritis, but more often the habit may exist for years without distinctive outward signs. A good color should be evident throughout the body, and the pink skin of health is too well known to require a description. *Vaso-motor contraction* produces local or general pallor as does an inefficient peripheral circulation. *A florid face* is common in gout, early interstitial nephritis

Cyanosis in ambulators.

Drug poisoning.

Addison's disease.

Argyria.

Sallow skin.

Syphilis.

Opium and cocaine habit.

May lack symptoms.

Normal color.

and hepatic cirrhosis, but may be due to idiosyncrasy, acne rosacea or exposure. It often suggests an over-luxurious and self-indulgent life or the abuse of alcoholics. *Unilateral flushing* is often observed in lobar pneumonia, or mere pillow pressure, migraine, and less commonly in irritation of the fibres of the cervical sympathetic as in aneurism of the aortic arch. As regards marked color loss or modification in connection with chronic diseases, it should be borne in mind that the symptom is as a rule one indicative of the *advanced stage* of the disease.

Unilateral flushing.

Bright's Disease.—Aside from the color changes referred to in the preceding paragraph, the external appearances in Bright's disease may be somewhat characteristic in chronic interstitial nephritis and yet more in chronic parenchymatous nephritis and active Bright's. In the first, the chief indication is found in the tendency to edema of the eye-lids, especially of the lower. In such the pale, puffy and almost translucent lid of early morning may later in the day have become shrunken and wrinkled. The condition cannot be considered as pathognomonic, but should be regarded as suggestive. In chronic parenchymatous and in "mixed" nephritis, the face may also appear more or less puffy, sallow or even fawn colored. The stages of edema will be discussed under another heading. In acute Bright's disease, the peculiar pasty white and the evidences of edema are distinctive.

Nephritis.

Baggy eyelids.

Fig. 2.—Bright's Disease. Wrinkled lids.

Not pathognomonic.

JAUNDICE.—(*Icterus.*) Jaundice is either *obstructive*, as in direct or indirect inflammation or obstruction of the common duct, or *toxæmic*, the latter form being often wrongly termed *hematogenous* as opposed to *hepatogenous*. Both types are essentially hepatic as regards the source of bile pigment, and "obstruction" and "diffusion" jaundice cover nearly every type. There are toxines which act directly upon the blood itself (within the hepatic tissues) or affect the hepatic cells. Poisoning by phosphorus, potassium chlorate, ether, chloroform, toluylendiamin, snake venom and arsenic are examples of one form, yellow fever, pyemia and malaria of another, while certain diseases are *characterized* by a marked associated jaundice. Such are Weil's disease and acute yellow atrophy (malignant jaundice). It occasionally complicates pneumonia, ulcerative endocarditis, syphilis and even influenza. The typical mild jaundice is seen in simple catarrh of the bile ducts. The term "obstructive" as usually applied covers an obstruction to

Hepatogenous vs. Hematogenous.

Obstructive jaundice.

the passage of bile from the liver to the intestines with resultant absorption into the general circulation. The causes are severe gastro-duodenitis, catarrhal conditions involving the duct, gall stone, or parasites in the common duct, pressure closure by tumor of that or adjacent structures, and very rarely by fecal accumulation, the pregnant uterus, or an abdominal aneurism. Strictly speaking all cases of jaundice are obstructive as even in the toxic form there is a high viscidity which favors absorption. The remarkable jaundice associated with mental shock or profound emotion is considered by Herter to be obstructive and is probably due to spasm and reversed peristalsis. In one such case coming under the author's notice profound jaundice followed a mere fit of anger. Urobilin icterus may occur early in hepatic cirrhosis or common jaundice and the substance appears in the urine.

Emotional jaundice.

Urobilin icterus.

Symptoms.—The skin, *ocular conjunctiva* and mucous membranes of the throat yield the best evidence, especially if subjected to pressure by a microscope slide or a tumbler, or merely blanched by finger pressure, *but even a marked discoloration may be invisible by artificial light*, though daylight shows a color which on the skin itself varies from a dim or brilliant yellow to a deep green or bronze (melasicterus), sometimes simulating Addison's disease. Two cases of this sort seen recently by the author were due to the last stages of cirrhosis of the liver and a failing heart associated with general anasarca. *The sweat and urine are discolored*, the latter often yielding the first evidence of the condition, the pulse is often slowed, the stools are pale gray, pasty and fetid, either constipation or diarrhœa may be present and there may be a troublesome pruritus. *A marked hemorrhagic tendency is shown in severe cases* and is of special interest to the surgeon. There may be marked mental depression or extreme irritability, and in certain cases convulsions, active delirium or a typhoid state, ending perhaps in coma. Such cases are most commonly associated with the acute infections and the pulse and respiration are markedly slowed. *True malignant jaundice* (acute yellow atrophy) is a rare disease of unknown causation* chiefly affecting women and often related to pregnancy, sometimes to violent emotion or shock (see p. 308).

Outward signs.

Daylight necessary.

Melasicterus.

Stools, sweat, and urine.

Surgical bearing

Icterus gravis.

Icterus Neonatorum.—A mild type is extremely common in newborn children, occurring during the first 24 or 48 hours and lasting for a week or two. It is benign and unimportant. The grave form is due to sepsis, usually of umbilical origin, syphilitic disease of the liver or

Mild vs. Fatal.

*But 250 cases are on record since the first observation by Ballonius in 1616 (Osler).

congenital absence of the ducts. It is frequently associated with hemorrhage from the navel and is a fatal disease.

DRY AND MOIST SKIN.—Hyperidrosis.—Sweating of the hands or feet may indicate idiosyncrasy, debility, or be an indication of neurasthenia and especially of sexual neuroses. Unilateral sweating, especially of the head or face, like pallor or cyanosis, occurs in pressure involvement of the sympathetic, certain migraines and neuralgias. Sweating of the head is especially common in rickets and of half the body in rare instances of hemiplegia. General sweating occurs as a critical phenomena in certain acute diseases, as an associated symptom in malarial fever or phthisis and persistently in acute rheumatism, collapse, severe pain, etc.

Localized vs. General sweating.

Disease association.

Anidrosis.—A dry skin is associated with diseases causing a profuse discharge of fluid by the bowels or kidneys and is a pronounced symptom in certain of the cachexias where the skin may be both dry and harsh, diabetes, chronic interstitial nephritis and carcinoma furnishing good examples.

Significance.

Qualitative Changes.—The yellow sweat of jaundice, the blue and brown, yellow, red or even hemorrhagic perspiration of hysteria, the disgusting odors of bromidrosis or the urinous odor of the sweating associated with impaired renal function may be encountered.

EDEMA.—Three terms are used in this connection, *edema proper*, confined to the actual connective tissues; *dropsy*, the accumulation of fluid in the serous cavities or these combined with edema; *anasarca*, (general edema). The last term however is very generally used to indicate a combination of the two. Edema represents an excess of transuded lymph over the absorptive capacity of the tissues and may be obstructive (passive congestions), hydræmic (toxæmias, infections, cachexias) or in many instances a combination of both. Increased permeability of the capillary walls is probably a prominent factor in the latter if not in both groups, and experiment fails to prove the older theory of hydræmic plethora, i.e. increased blood volume and water retention. The distinction between inflammatory edema and ordinary edema is largely dependent upon the physical constitutents of the inflammatory *exudate* as compared with the simpler *transudate*.

Obstructive vs. Hydræmic.

Inflammatory.

A thorough knowledge of the favorite locations and the vagaries of this condition is absolutely essential, inasmuch as it may shift its seat, obey or disobey the law of gravitation, and vary from swelling of the lids and slight puffiness of the ankles, or a localized angio-neurotic edema, to that distressing condition known as general anasarca, in which the

Full of vagaries.

Variable in degree.

patient is drowning in his own secretions. The term, *angio-neurotic edema* is applied to a localized and transient firm swelling closely allied to urticaria and erythema nodosum, not ordinarily pitting on pressure, found in various regions of the body in certain ill defined conditions, and is usually of but slight interest or importance. *Slight and transient edema* of the lower extremities is found in cases of anæmia, or after prolonged and exhausting tramps in those unaccustomed to physical exercise. The characteristic feature of all edemas is the appearance of *a swelling* due to fluid in the connective tissue spaces, *tending to destroy the normal outline* of the affected portion, readily *receiving and* for a variable period *retaining indentations from pressure* of the examiner's finger or constriction due to clothing. In its extreme grades the same process *tends to produce liquid exudate in such serous cavities* as the pleura, pericardium, tunica vaginalis and peritoneum, producing the condition known respectively as *hydrothorax, hydropericardium and ascites.* Such extreme effusions accompanying general edema constitute "general anasarca," which in practice is limited to Bright's disease, or a failing heart. As between these two conditions the attendant edema at times presents some distinguishing features; in heart disease general edema (predominantly obstructive) even without general anasarca is associated with cyanosis and in its original seat and progress usually follows the law of gravity, commencing in the feet and extending upwards. In acute parenchymatous nephritis any marked edema (hydræmic) usually appears first in the face and eyes and extends downwards, being associated with marked pallor, unmixed with any considerable degree of cyanosis, unless there be obstructive effusion in the peritoneal cavity or thorax, or a complicating failure on the cardiac side. Furthermore, the edema of Bright's disease as it appears on the body generally or the extremities, is distinctly firmer than is a *recent* cardiac edema, and in those with a delicate skin, more especially in children, one may find that the legs present a marble appearance, the blue superficial venules contrasting with the dead white of the skin. Edema of the cardiac or renal type is markedly affected by changes in the position of the patient, that of heart disease especially so, the attitude assumed by such a case during any night being often indicated in the morning by an increased swelling of the side upon which he has lain. Cardiac edema usually appears first in the most dependent portion of the body, as in the feet and ankles after a day's activity, or over the sacrum in the bedfast. The slighter edemas of the lower extremities may wholly or in part disappear during a night's

Angio-neurotic form.

Salient points.

Cardinal signs.

Cavity exudates.

General anasarca.

Cardiac edema.

Renal edema.

Renal lacks cyanosis.

Marble edema.

Effect of attitude.

Effect of exercise.

rest in bed. A certain form of hard edema is occasionally met with in those curious cases in which the patient is able to be about for weeks, months or years, in spite of a chronic edema of the lower extremities, often unrecognized. The skin becomes leathery and pigmented because of recurrent eczema, and so resistant that *prolonged firm pressure is necessary to indent it.* One should not be misled by an edema of the lower extremities, more often unilateral, due to varicose veins. Edema of the calves without edema of the ankle or other regions has been observed by the author in several cases of heart disease.

Leathery edema.

Varicose veins.

Important variation.

Collateral Localized Edema.—Over all purulent exudates or suppurative inflammation such edema *may, but does not always occur*, as for example, in empyema, mastoiditis, parotitis, pericarditis, hepatic abscess, perinephritic abscess, etc. Non-inflammatory circumscribed edemas are either angio-neurotic purpurie, giant urticarial or the slight edema of the ordinary obstructive type, bilateral if of the cardiac or renal type, unilateral if due to the blocking of the local circulation. Edema of the arm may be due to the pressure of tumors or enlarged glands, to thrombosis and even to massive pleuritic effusion or mediastinal tumors including aneurism. Similar causes will account for local edemas of the lower extremities.

Associated conditions.

ASCITES.—Fluid in the peritoneal cavity may be part of a general dropsy as in heart disease, Bright's disease or obstructive pulmonary conditions such as emphysema and fibroid lung, or it may result from any form of chronic peritonitis, whether simple, tuberculous, or due to tumors, malignant or otherwise. Furthermore, it may be caused by portal obstruction, whether due to disease of the liver itself, as in cirrhosis, or to thrombosis or pressure. In tuberculosis, malignant disease and most of the cases associated with new growths the effusion is relatively small. Occurring as a part of general dropsy or portal obstruction, the exudate is ultimately large and produces a tense protuberant abdomen *which tends to broaden in the flanks if the patient assumes a dorsal recumbent position.* The *umbilicus is prominent, percussion reveals a dulness dependent upon gravity, and hence shifting as the position of the patient is changed.* The fluid seeks the most dependent portion, the intestines float upward and yield a tympanitic note, *shifting exactly as does the dulness but being diametrically opposite in position.* The patient being in a sitting or standing posture a *fluctuation wave* is felt if the abdomen is sharply tapped upon one side just below the line of percussion dulness, while the fingertips of the other hand are placed opposite. The ulnar surface of the hand of an assistant should be

Small effusions.

Large effusions.

Appearance.

Shifting dulness.

Ascitic wave.

firmly applied in the median line between the percussing and receiving fingers to interrupt a false vibration in the wall otherwise indistinguishable from the true ascitic wave. Shifting dulness may be interfered with by adhesions which prevent the flow of the exudate or hold intestinal coils in a fixed relation to the wall; moreover, some time may be required for the fluid to change its site, and several moments should be allowed to elapse before negative findings are reported. Such effusion makes palpation of the spleen, liver or underlying tumors difficult and demands that the palpating finger shall be suddenly depressed, preferably both during forced inspiration and at the end of forced expiration, the temporary displacement of the underlying fluid sometimes making the underlying structure palpable.*

False wave.

Sources of error.

Dipping.

The *knee elbow* position is necessary for the detection of small exudates, the dulness then appearing in the very region ordinarily most resonant. In massive effusions with excessive tension the ascitic thrill or wave may be *but rarely is* absent.

Small effusions.

The elaborate tables of differential diagnosis seem to the author almost futile. Movable percussion dulness in the flanks and lateral dulness with central resonance at once rules out *pregnancy* and *pancreatic*, *ovarian* or *hydatid cysts*, the signs being diametrically opposite. Encysted ascites may give rise to insuperable diagnostic difficulties. *Meteorism*, i.e. excessive tympanites yields a universally tympanitic note. The ascites of portal obstruction, usually due to hepatic cirrhosis, stands by itself, though in any massive accumulation a variable degree of edema may be present in the lower extremities. Such an ascites without general edema co-exists frequently with emaciation, giving rise to the chief form of "poached egg belly." A big belly in a thin man invariably means a pathologic condition.

Differentiation.

Slight or no edema in cirrhosis.

Poached egg belly.

Character of Ascitic Fluid.—It is usually clear, straw colored, of low specific gravity (1010-1015), is albuminous and may spontaneously coagulate. The color is slightly darker in cirrhosis than in the ordinary secondary forms. In tuberculosis and malignant disease it may be hemorrhagic and rarely it is milky or turbid from fat or true chyle. The two latter conditions are readily distinguished by the size of the fat globules which in chyle resemble a closely packed field of cocci as shown on microscopic examination. Mere blocking of the thoracic duct may or may not cause chylous ascites; rupture may occur or filaria sanguinis hominis be present.

Color.

Chylous vs. Fatty ascites.

*As such cases almost invariably need to be tapped the manœuvre is of slight practical use.

Crepitation of skin.

SUBCUTANEOUS EMPHYSEMÆ.—Aside from malignant edema and glanders this condition indicates the entrance of air into the subcutaneous tissue through wounding or rupture of an air containing viscus, as in tracheotomy, cough, rupture of pulmonary alveoli, malignant ulceration of the esophagus, etc. It offers no difficulty in diagnosis because of the pathognomonic crepitation of the distended tissue under finger pressure.

SUBCUTANEOUS HEMORRHAGES.—These will be fully considered under purpura, and it need only be said that the discoloration of the effused blood is persistent under pressure, that the spots undergo the same changes in color as an ordinary bruise, are not elevated, and vary greatly in size. The term ecchymosis covers the ordinary petechia in the smallest type. When complicating an exanthem as in measles or smallpox, they invariably indicate a severe type of disease. In chronic diseases they are ordinarily associated with a definite cachexia or a hemorrhagic tendency, (see purpura, page 410). In certain obscure infections they point to sepsis and if endocardial murmurs are present to septic endocarditis. They should not be confused with the nodular swelling of erythema nodosum.

Petechiæ and ecchymoses.

Erythema nodosum.

COLLATERAL VENOUS CIRCULATION.—Hepatic cirrhosis or thrombosis of the portal veins may produce a very marked and evident enlargement of the superficial abdominal veins, and a similar condition occurs in thrombosis of the inferior vena cava. The former chiefly affects the median region of the abdomen; the relation of its veins to the navel suggesting the term "caput medusæ," and though the lower thoracic veins are involved the whole group lies chiefly within lines dropped from the middle of the clavicle to the groin. In the latter the enlargement is predominantly lateral and usually less complicated in pattern. Unfortunately, admixture of the two may occur as in the case of a massive ascites of cirrhosis producing caval obstruction leading to enlargement of both groups. If the valves are competent the direction of blood flow is easily determined by stroking the blood from a vein and making compression. In any form of intra-thoracic tumor, especially those of the mediastinum, visible enlargement of the internal mammary and intercostal veins may be evident if the big trunks are compressed. Obliteration of the descending aorta at the ductus arteriosus is a clinical curiosity, the child usually but not always showing a collateral arterial circulation between the subclavian above and the vessels of the lower extremity. The arborescent branchings, indicating the diaphragmatic attachment frequently observed on the walls of the chest, seem to be

Portal obstruction.

Caput medusæ.

Vena cava.

Internal mammary and intercostals.

Visible arteries.

without definite clinical significance. The author's observation would indicate that they are most frequently observed in cases showing obstructed pulmonary circulation of the chronic type, in chronic cough, and not infrequently in connection with general arterial or myocardial degeneration. Yet they may exist in the normal individual. Lower thoracic veining.

DRUG ERUPTIONS.—Although the various exanthems will be considered under the related diseases, the question of drug eruption needs separate discussion. The iodides may produce acne-like or even varioliform or erythematous rashes, the bromides an acne-like eruption, while phenacetin and its congeners, the balsams (such as copaiba), sodium salicylate, diphtheria-antitoxin and various other sera are capable of producing urticaria or rashes the more exactly simulating scarlatina or measles, as fever *may be coincident.* Hundreds of drugs produce rashes in certain individuals and these efflorescences are of so varied a nature as to preclude farther description.* Simulate many diseases.

DESQUAMATIONS.—The chief diseases followed by desquamation are:—scarlet fever (lamellæ), measles (bran-like scales), smallpox (crusts), erysipelas (flakes), dermatitis (exfoliation).

SCARS.—*Study the history of scars.* The pits of smallpox may exclude that disease in the case of a doubtful exanthem. The scar of a carbuncle at the back of the neck may suggest glycosuria, multiple linear scars at the angles of the mouth congenital syphilis. Linear scars on the left hand usually indicate right-handedness and vice versa. On the chest small bright shining slightly depressed scars may indicate the past use of croton oil, and grouped, finely linear, parallel scars over the lungs, liver, heart or spleen suggest cupping for the relief of some pain or acute inflammation. Scars at the waist line are common in those who have worn electric belts. Those in the supraorbital region or thoracic zones may suggest supraorbital or pectoral Zoster. The *tiny depressed scars* of acne affect the chest, shoulders or face and more generally distributed suggest syphilis. Those over the heel and scapula suggest past bed sores and hence past severe ailments such as typhoid. Bullet and stab wounds† may be suggestive in relation to character, past occupation and present disease. A seton may have left its double scar on back of the neck of some middle aged or elderly man. Scars about the neck if long and linear and particularly if left sided Important inferences. Cupping. Electric belts. Bed sores. Gunshot and stab wounds. Suicide.

* For an excellent discussion of this topic see Allbutt's System of Medicine.

† In a clerical appearing patient from a neighboring state such a wound proved to have been the result of a drunken brawl in a bawdy house and led to an investigation of past and present habits that proved illuminating.

in origin and transverse suggest suicidal attempts. They usually run downward from the angle of the jaw. Puckered, depressed, irregular scars in the cervical triangles, especially the anterior, suggest the slow healing of broken down tuberculous glands. Scars evidently due to surgical operations usually indicate by their position and extent the nature of the operation and its probable cause. Scars in the groin do *not* suggest past syphilis, but rather chancroid. Indeed there is not much of positive differential or diagnostic value in syphilitic scars save in certain situations or in certain forms of the eruption. In general one may say that syphilitic scars may be round, reniform, oval or horseshoe-shaped, smooth and seldom traversed by fibrous bands except at joints. Lupus may closely simulate syphilis but is not multiform. Frequently there are multiple punctate depressions and some old luetic scars are pliable and have a brownish red areola. (See also syphilis.) Of special significance are the scars indicating a primary lesion, usually, of course, upon the genitals, and those of a destructive ulceration of the soft palate. The scar of operation for epithelioma of the lips or nose is especially suggestive. Bluish marks from old needle coated with oxide of iron may suggest confirmed morphinism or cocainism.

Tuberculosis.

Chancroid not chancre.

Lues.

Epithelioma.

Hypodermic marks.

THE HEAD.—Open sutures persisting after the 9th month suggest hydrocephalus, cretinism or rickets; the posterior fontanelle should be closed at the end of the second month, the anterior at the end of the second year; delayed closure or unusual size suggests hereditary syphilis, rickets, and cretinism. Bulging fontanelles occur in chronic hydrocephalus, meningeal hemorrhage or inflammation; slight prominence and pulsation may occur in any child suffering from febrile ailments and is negligible. A sunken fontanelle is present in wasting disease and spurious hydrocephalus.

Sutures and fontanelles.

Cranio-Tabes is indicated by a soft crackle produced by pressure over circumscribed areas of the occipital or posterior portion of the parietal bones and indicates rickets or syphilis. *The pulsating congenital tumors* of variable size bulging from the sutures are classified as hydrencephalocele, meningocele or encephalocele. *Wens or cysts* are common in the scalp and should not be confused with the deeper seated immovable syphilitic nodes representing a gummatous periostitis; co-existing brain symptoms suggest similar growths on the inner surface, their consistence is soft and doughy and there are nocturnal exacerbations of the pain.

Tumors and nodes.

Rickets.—A flattened head with elongated vertex, high, square,

often frontally projecting forehead surmounting a small face indicates rickets and is usually associated with delayed closure of the fontanelles, a sweating forehead, muscular weakness, backwardness, delayed growth, beading of the costo-chondral articulations ("rickety rosary") especially the 5th and 6th, epiphyseal swellings and a palpable thinning of the postero-inferior aspect of the parietal and occipital bones (cranio-tabes). Deformities of the chest, legs and spine are common and the restless little sleeper may have rubbed away the hair at the occiput. Caput quadratum. Rickety rosary.

Hydrocephalus.—If the child's head be large but globular rather than square, the face relatively small, the space between the eyebrows prominent, the fontanelle bulging, the sutures separated and the external veins visibly distended a history of excessive cranial growth or congenital deformity will be obtained and hydrocephalus is evident.

Osteitis Deformans.—A marked increase of the head circumference without facial involvement may be associated with the *curvature* of the *enlarged* shafts of the long bones characterizing the disease.

Leontiasis Ossea.—Frequently produces marked deformity of the skull, particularly in the frontal regions, by osteophytic deposit.

Facial Hemiatrophy.—Slight unilateral differences may constitute degenerative stigmata but in true hemiatrophy the face is mesially divided into distinctly different halves, the extreme and apparent emaciation of the one side contrasting sharply with the better nourishment of the other. A curious contrast.

THE EYELIDS.—*Dark circles or duskiness most* marked in brunettes are common indications of menstrual disturbance or pelvic disease. In both sexes and at any age they may indicate exhaustion from pain, insomnia, over exertion, weak heart and exhausting diseases. *Puffiness or swelling* of the lids may indicate Bright's disease, arsenical poisoning, anæmia or pertussis. Unilateral swelling may be due to angio-neurotic edema or actual inflammations, such as boils, erysipelas or glanders. Unilateral swelling with exophthalmos may indicate cerebral thrombosis or tumor pressure. Inflammation of the lids themselves may indicate conjunctivitis, simple or specific, and is a common manifestation in measles, coryza, iodism, hay fever and eyestrain. Styes, chalazion, warts, epitheliomatous or syphilitic ulcers, gouty tophi, lacrymal cyst or obstruction and blepharitis marginalis may be evident but need no extended description. Trivial or important. Unilateral vs. Bilateral.

THE EYES.—Many valuable inferences may be drawn from careful inspection of the eye and the expression may indicate bad habits con- Concealed vices.

cealed, and often gives a clue to the whole temperament and disposition of the patient. The author has been especially interested in the study

Insanity.

of the eyes of the insane, or those of insane hereditary predisposition. Study of the background is of course a matter for special investiga-

Ocular palsies.

tion (see p. 557). Paralysis of the ocular muscles as indicated by ptosis or strabismus at once suggests *syphilis*, brain tumors, locomotor ataxia, cerebral hemorrhage, or in acute diseases, meningitis or profound toxæmia (see also p. 560)

Sometimes trivial.

Nystagmus is an involuntary lateral, rotatory or vertical movement of the eyeball, and *in the absence of extreme refractive errors* suggests disseminated sclerosis, tumors of the cerebellum or pons, locomotor ataxia, or more rarely Friedreich's ataxia or epilepsy.

Of little account.

The Arcus Senilis.—This is a symptom of little significance when seen in persons beyond middle age, as it is merely a local indication of the general degenerative process; in younger persons it is of much more significance and may be associated with symptoms of general vascular changes. It is indicated by a white line surrounding wholly or in part the corneal margin.

Bilateral vs. Unilateral.

Exophthalmos.—In most cases this is bilateral and associated with exophthalmic goitre, both eyes being unduly prominent and producing a peculiar staring expression, striking and easily recognized. Unilateral exophthalmos usually indicates a growth or tumor of some kind in close relation to the affected eye.

Enophthalmos.—This is the opposite of exophthalmos and ordinarily accompanies wasting diseases, severe hemorrhage or profuse diarrhœas. Unilaterally it is found in hemiatrophy or lesions of the sympathetic causing malnutrition.

Fig. 3.—Exophthalmic Goitre

Corneal Opacities.—These indicate as a rule syphilis or a tuberculous tendency but they may result from injuries of any nature.

Cataract.—In many cases this disease of the crystalline lens seems to reflect a general degenerative process, and should suggest a painstaking investigation of the heart, blood vessels and kidneys. Juvenile cataract is frequently associated with struma or congenital syphilis.

The Dry and Moist Eye.—If the eye be permanently or for long uncovered by the lids, it becomes glazed and dry as also in profound

collapse. On the other hand excessive lachrymation may accompany irritation of the conjunctiva or photophobia and actual overflow be present if the lachrymal duct is blocked or displaced (epiphora).

THE NOSE.—Saddle nose is discussed elsewhere; a coarse, broad organ is seen in certain strumous types, and in cretinism, or may be purely racial. Its base may be broad and shapeless in adenoid disease and nasal polypi and it may be the seat of distressing vascularity even in the temperate individual. Such innocent redness is most frequently due to exposure to the elements, gastric and pelvic disturbances or chronic obstructive nasal catarrh. The alæ may carry the tuberculous or epitheliomatous ulcer.

Misleading redness.

Sneezing needs no special discussion though occasionally a troublesome phenomenon.*

Working Alæ.—In neurotic high strung individuals this is commonly observed and it also constitutes one of the signs of dyspnœa.

THE EAR.—Earache.—In infants the condition may be indicated only by crying, restless movement of the head or rubbing the ear. In them as in profoundly toxæmic adults (typhoid, etc.) an acute suppurative otitis media may be entirely overlooked until the discharge appears. The condition is a common one in acute infections (especially scarlet fever, influenza, typhoid, diphtheria and measles) and its complications are so serious (mastoiditis, meningitis, brain abscess, etc.) as to demand careful watching in such cases. Earache may also be due to impacted cerumen, abscess or ordinary furuncle of the meatus externus, simple catarrhal inflammation and blocking of the eustuchian tube, decayed teeth and alveolar abscess, foreign bodies, neuralgia, and rarely cancer of the tongue or even innominate aneurism.

Often overlooked.

Hematoma Auris.—Bruising and swelling of the pinna is frequently encountered in the insane.

Cyanosis and Anæmia are well shown by the external ear. It is the most frequent situation for frost bite—and may be the seat of ochronosis (blue black cartilages), persistent localized gangrene,† gouty tophi (gritty sodium urate nodules near the margin of the pinna).

Ochronosis, gangrene and tophi.

Discharges of various kinds may be noted. Slight and serous if due to meatal eczema, purulent if from purulent otitis media or meatal abscess—clear and serous or primarily bloody in fractures at the

*In one instance known to the author persistent sneezing excited by normal sexual stimuli proved absolutely intractable.

†In a case of drug habituation a recurrent gangrene of years duration affected the ear alone.

base of the skull—hemorrhage from the lobe is not uncommon in hemophilia even without wound or abrasion. Air pressure as in artillery concussion or caisson disease may produce hemorrhage. (See also auditory nerve and its diseases, p. 564.)

Herpes.

THE LIPS.—The grouped vesicles of herpes common in coryza, pneumonia, malarial fever; the initial lesion of syphilis or the mucous patches of the same disease, epithelioma and fissures are some of the important conditions to be noted. Epithelioma usually occurs at or beyond middle age, ordinarily affects the lower lip, grows indolently and exists for a long time without marked involvement of the neighboring glands. The initial lesion of syphilis is *promptly* followed by glandular swelling. Mucous patches and syphilitic fissures tend to involve the angles of the mouth and leave linear radiating scars, but ordinary "cold cracks" or fissures are of no significance. Acute swelling of the lips commonly indicates trauma, insect bite, angio-neurotic edema, simple abscess, or more rarely corrosive poison, cancrum oris, erysipelas or phlegmon. Aside from paralysis or primary mental defects the dry lips and open mouth are seen in coma, chronic hypertrophy of the tonsils or a nasal obstruction (usually due to adenoids), inflammation of the buccal cavity, the profound exhaustion of certain acute adynamic febrile conditions and the terminal stages of chronic disease. The lips are primary indicators of anæmia and cyanosis.

Epithelioma.

Syphilis.

Acute swellings.

Mouth breathing.

Pallor and cyanosis

Poisoning.

The Odor of the Breath.—In acute poisoning this may yield information of the first importance and the odor of carbolic acid, the aromatic bitter-almond odor of hydrocyanic acid and the peculiar smell of ether, chloroform, laudanum and alcohol are readily detected. In the terminal stages of diabetes mellitus the fragrant fruity breath is most suggestive and of serious import. Phosphorous yields an unmistakable odor. Foul breath in itself most often indicates improper care of the teeth, constipation, chronic naso-pharyngeal catarrh or local disease of the buccal pharyngeal or more often the naso-pharynx. The bad breath of chronic drinkers is usually due to chronic gastritis. A peculiar heavy, aromatic odor is noticeable in so-called uræmia or in the terminal stages of failing heart, and a peculiar cadaveric emanation is sometimes noticeable at the time of death from exhausting disease.

Diabetes.

Phosphorus.

Respiratory and alimentary tracts.

Uræmia.

Cadaveric emanation.

THE BUCCAL CAVITY.—The pigmented areas of Addison's disease appear here as on the tongue, as do jaundice and cyanosis. In acute exanthematous diseases it may be the site of the earliest eruption, as in measles where small red spots with a tiny bluish white center appear before the development of the cutaneous rash (Koplik's spots). The

Pigmentation.

Exanthems.

papules and vesicles of varicella and variola may be seen and the vivid redness of scarlet fever is characteristic. As regards secretion, there may be either *dryness*, such as occurs in mouth breathers from whatever cause, or *salivation* such as accompanies certain of the acute inflammations or results from the over-use of mercury. Either condition may be associated with the hysterical or neurasthenic states, and the drooling such as occurs in bulbar palsy, facial paralysis, diphtheric paralysis and idiocy is not necessarily attended by actual increase in secretion.

Dryness and salivation.

Hysteria.

STOMATITIS.—Inflammation of the buccal membrane may take the form of (a). **Simple catarrh** as seen in teething children, acute infectious disease, or as a result of gastro-intestinal disturbances or direct irritation. The condition is characterized by redness, dryness and heat of the mucous surface with increase of saliva, discomfort upon taking food and moderate fever.

Teething children.

(b). **Follicular or Aphthous Stomatitis.**—Is an evidence of impaired general health and digestion, common in children, not infrequent in adults, and is characterized by small vesicles soon transformed into superficial small ulcers with an inflamed areola, often appearing in crops and involving chiefly the edges and the tip of the tongue, the deeper folds, or the inner aspect of the cheeks. Constitutional symptoms are identical with those of the catarrhal form but the process may be persistent or spread and give rise to a greater degree of pain and irritation.

Indigestion and malnutrition.

Seat.

(c). **Parasitic Stomatitis.**—(Thrush, muguet, soor, mycotic stomatitis). This is due to the *oidium albicans* shown readily under the microscope. It occurs chiefly under conditions of poor nutrition and uncleanly artificial feeding in infants, and is characterized by small curd-like deposits appearing first upon the tongue, but tending to coalesce spread and even, in extreme cases, cover the whole aural and pharyngeal surface. The patches are readily removed and leave no ulceration or excoriated surface. The diagnosis depends upon the primary location of the lesions, their peculiar superficial nature and the use of the microscope.

Oidium albicans.

Curd-like deposits.

Mercurial Stomatitis.—Either through occupational poisoning, idiosyncrasy or over dosing, mercury may cause profuse salivation associated with spongy swelling and even ulceration of the gums or rarely necrosis of the jaw. A metallic taste and fetid breath with tenderness of the teeth are usually the first symptoms, and patients under mercurial treatment should be carefully watched for increased flow of

Salivation.

First symptoms.

saliva, and made to bring the teeth sharply together at each visit in order that tenderness may be detected.

Rare. In slums chiefly. Symptoms. Prognosis.

Gangrenous Stomatitis. (*Noma.*)—Fortunately this terrible ailment is rare, being seldom encountered save in the badly nourished and environed children of the slums, in whom it follows convalescence from acute fevers, chiefly measles. It is characterized by the appearance of a sloughing, spreading ulcer usually on the cheek, rarely on the gum. The breath is fetid, there is high fever, profound prostration, delirium and diarrhœa. Gangrene rapidly develops in the cheek and may involve adjacent structures to a frightful degree, or appear in other portions of the body. The disease is almost invariably fatal.

Severe type. Seat. Prognosis.

Ulcerative or Fetid Stomatitis.—This condition stands midway between the aphthous form and cancrum oris, occurring during dentition usually; but rarely in adults, and at times seeming to be epidemic. The gums are usually swollen and spongy, bleed readily and show linear sloughing ulceration. The process occasionally involves the tongue, cheeks and inner surface of the lips; the submaxillary glands enlarge and the constitutional symptoms are often severe and associated with extreme prostration and marked febrile reaction. Rarely, and chiefly in marasmic children, the process assumes a type suggesting noma, but ordinarily recovery occurs in a week or ten days.

Coated tongue. Gastric ulcer. Typhoid tongue. Beefy tongue Strawberry tongue.

THE TONGUE.—In many diseases, acute or chronic, whether affecting proximate or remote structures the condition of the tongue may assist diagnosis, prognosis and treatment. A *lightly coated* tongue may be found in persons of perfect health, or may indicate fever, disease of the naso-pharynx, gastro-intestinal disturbances, mere smoking, drinking, or the possession of bad teeth. A *heavy pasty yellow coating* in usually indicative of catarrhal gastritis, naso-pharyngeal catarrh or some disturbances of the digestive tract. A *clean tongue* may be and often is associated with gastric hyperacidity or even ulcer. *In chronic dyspepsias* the tongue is *flabby, often enlarged and laterally indented by the teeth;* in the *typhoid state the heavily coated tongue* is ordinarily bright red along its margin and at its tip, which oftentimes shows a triangular red area. In all conditions of *profound toxæmia* and *exhaustion* it tends to become *brown, dry* and *fissured,* is *tremulous, slowly protruded and retracted slowly* or only when ordered. Such a tongue and its congener, the *red, shiny, dry, "beefy"* tongue indicates a bad prognosis. The so-called *strawberry tongue* is frequently seen early in an attack of scarlet fever. Its name is derived from its bright red surface-points due to swollen papillæ emphasized by a white pasty

background. A *unilateral coating* suggests hemiplegia, but may be due to bad teeth on the affected side. *Deep fissures* along the edge suggest past syphilis; it reflects the general pallor of anæmia and the cyanosis of cardiac or pulmonary disease, and one may detect a tuberculosis or epitheliomatous ulceration or scars suggestive of the tongue biting of epileptic seizures or glosso-labio-pharyngeal paralysis. *Unilateral atrophy*, the *tremor* and *jerky protrusion* of marked alcoholism or actual delirium tremens and muscle spasm, associated with paresis must not be forgotten. *Marked enlargement* may be due to acute glossitis or to acromegaly and myxœdema. *Tumors* must be thought of as well as the various acute conditions affecting the mucous membrane of the mouth. Various *pigment deposits* may occur here as in other portions of the oral mucous membrane, the most marked are the deep brown, purple or black deposits of Addison's disease, and the yellow tint of the under surface in jaundice. A *black tongue* usually indicates the use of iron, bismuth or charcoal, the *brown discolorations* are most commonly due to the use of tobacco, liquorice or chocolate, less often to laudanum and rhubarb. The corrosive acids either whiten the surface (oxalic, carbolic, sulphuric acids as well as ammonia and corrosive sublimate) or yellow it as do chromic, hydrochloric, and nitric acids, while caustic soda or potash and the nitrate of mercury redden it. It may be the source of persistent hemorrhage in purpura or hemophilia. The term "geographic tongue" is applied to the annular red patches due to eczema, which spread peripherally to form eccentric areas.

Fissures.

Scars.

Tremor and atrophy.

Large tongue.

Pigment.

Black tongue.

Poisoning.

Hemorrhage.

Eczema.

Leucoplakia Buccalis.—(Buccal psoriasis, leucoma, ichthyosis). Is characterized by non-ulcerative and irregular smooth, white, firm raised patches. The *smoker's patch* occupies the dorsal surface of the tip as a pearly yellow, or perhaps reddish plaque.

Smoker's tongue.

The Gums.—Marginal redness is usually of little significance, being attributable to uncleanliness, and dental caries, though it is thought to have some significance in young persons as an indication of tuberculosis and is present in several of the cachetic states. A *grayish or bluish gray or black line* may indicate lead poisoning but this line is rather a linear series of dots and lines about one mm. from the actual margin. Cabot justly protests against the use of the term "*blue line*" in these cases. More rarely a similar *greenish blue line* is present from copper poisoning. *Swollen, spongy or bleeding gums* are present most commonly in mercurial stomatitis and conditions of great debility and exhaustion. In scurvy it constitutes one of the prominent symptoms and is present oftentimes in the various cachexias. In neglected cases

A misnomer.

of the typhoid type accumulation of oral debris from the so-called *sordes*. Dental abscesses and various growths such as epulis may also occur in this region. Around the base of the gums as well as in the more readily visible portions of the buccal mucous membrane may be seen the *mucous patches of syphilis*.

The Teeth.—As regards the period of eruption of the first and second sets of teeth, a wide margin must be allowed in both directions, an unusually early appearance being of no significance while a *greatly* delayed eruption is usually indicative of malnutrition and often associated with distinct developmental defects. The disturbances of dentition while doubtless exaggerated are nevertheless important and are accountable for many ailments of infancy. Any doubting physician would be convinced of this fact if he himself should for a time suffer from similar inflammations from whatever cause. On the other hand he should not too readily adopt the disturbance of dentition as a cause of disease lest he overlook more concrete and serious conditions.*

Hutchinsonian Teeth.—These teeth, characteristic of congenital syphilis, are often more or less closely simulated and a positive diagnosis should be made only when they present the following typical characteristics:—In the second dentition, peg-like, rounded, upper central incisors usually discolored, irregularly placed, separated and bearing a single well defined notch at their cutting edge or even less characteristic teeth may be termed syphilitic if associated with a history of middle ear disease, keratitis, sore bottom, and fissures or residual linear scars at the angles of the mouth. Transverse grooves or furrows like those of the nails indicate past severe illness, dentated cutting margins are common in malnutrition and pitted teeth may be due to stomatitis. Early decay is common in pregnancy, phosphorous poisoning, rickets, diabetes and other forms of severe malnutrition and the importance of good teeth in connection with chronic dyspepsia is too little appreciated by the practitioner. Many obstinate and persistent ailments are promptly cured by proper attention to the remaining teeth, the use of dental bridges, or extraction and the substitution of properly fitting plates.

* The normal periods are in months:—*First dentition:*—two lower central incisors 6–9. Four upper incisors 8–12. Four anterior molars and lower *lateral* incisors 12–15. Canines 18–24. Posterior molars 24–30 (in all 20 teeth). *Permanent set* (*in years*):—*Molars*, first set 6; second set 12–15; third set 17–25. *Incisors* 7–8. *Bicuspids* 9–10. Canines 12–14. It follows that at the end of the first year there are 6 teeth; at the end of the second 16, and that the first dentition should be completed in two and one-half years.

Teeth grinding is a common symptom of reflex irritation or gastric disturbance in children and is rarely due to the commonly accepted cause, worms.

The Jaws.—The high arched palate so commonly associated with the neurotic temperament is readily noted as is the massive lower jaw of acromegaly. The prominent square jaw and the receding chin are universally associated with firmness or the lack of it, but the conclusions are subject to doubt or at least to many exceptions. Spasm of the jaw suggests hysteria, tetanus, strychnine poisoning, trismus neonatorum, reflex spasm in childhood or irritation of cerebral origin. Its opposite, paralysis may be noted in pontine hemorrhage, neuritis, brain tumor, meningitis, etc. The jaw movement may be more or less impeded in such diseases as quinsy, mumps, and trichiniasis.

Fig. 4.
1. Heberden's nodes.
2. Syphilitic dactylitis.
3. Clubbed fingers.
4. Arthritis deformans.
5. Spade hand of acromegaly.

The Soft Palate.—Syphilitic perforations have been referred to. Paralysis is usually due to diphtheria, less commonly to cerebral tumor, meningitis, caries and bulbar palsy. Perforation and paralysis.

THE HANDS.—Aside from the stigmata of occupation, the hands afford much information of the most suggestive sort. The handshake itself may indicate a lack of vigor; tremor may often be felt as well as seen and what seems like a mere formality may contribute largely to diagnosis and prognosis. Persons of the tubercular type often have hands, delicate and feminine, easily compressed and weak in grasp and the flexible slender hand of the nervous person, sharply contrasts with the clumsier one of the phlegmatic type. In wasting diseases the hand feels hot, dry and flabby and emaciation is easily recognized, as are deformities of the joints due to chronic rheumatism, gout or rheumatoid arthritis, the clubbing of the finger tips of chronic pulmonary disease and the giant hand of acromegaly. The handshake. The grip. Wasting disease. Deformities.

Finger Nails.—*Chronic* indolent ulceration surrounding the nail suggests syphilis or marked malnutrition. *The incurved nail* associated with a clubbed finger tip is a sign of chronic pulmonary or cardiac disease of the obstructive type, or an advanced pulmonary tuberculosis. Onychia. Clubbed fingers.

Brittle or split nails.

Transverse ridges.

Brittle, striated or split nails most frequently occur in connection with gout, peripheral neuritis, and prolonged exposure to the X-Ray. Most important of all to the case-taker are the transverse ridges indicating a past severe illness. These are easily noted, last for a period of over 6 or 8 months, and by their proximity to the matrix indicate approximately the date of the illness.

THE ARM AND LEG.—Many of the conditions affecting these extremities such as edema, pain and tenderness have been discussed under their appropriate headings. Hard circumscribed bilateral swellings upon the ulna or tibia are usually syphilitic nodes, and the more diffuse, *bilateral*, painful enlargements of the tibia with nocturnal exacerbation are also usually syphilitic. *Localized edema with heat and redness* and severe deep seated pain over long bones and especially the tibia suggest acute periostitis which may lead to chronic fistulous openings indicating secondary necrosis. Enlargement of the lower end of the radius suggests rachitis, and exquisitely tender brawny induration over the femur in children is an almost constant sign of scurvy. Varicose veins are common in the lower extremities and may be bilateral or unilateral, are often associated in middle aged or elderly persons with chronic indolent ulcers and may produce a misleading edema. Congestion and edema with pain and localized tenderness suggest phlebitis, and perforating ulcer usually under the ball of the great toe may be present in locomotor ataxia or rarely in diabetes. Deep cyanosis of the foot or toes suggests Raynaud's disease, frost bite or the early stage of an *actual gangrene* which may be associated with injury, arterio-sclerosis, embolism, diabetes, frost bite, ergotism, Raynaud's disease and rarely, it is claimed, exophthalmic goitre. Mottled, dusky redness and pain over the sole of the foot may indicate erythromelalgia. The presence or absence of pulsation should be carefully noted in connection with painful diseases of the lower extremities especially as indicating arteritis or incipient gangrene. The enlarged glands in the groin or femoral triangle suggestive of syphilis, chancroid, Hodgkin's disease, leukemia, malignant disease, etc., should be noted as well as the enlarged epitrochlear gland of syphilis above the inner condyle of the humerus. *Transient nodular swellings* usually painless and subsiding with bruise like color changes are common in erythema nodosum. The region of the hernial opening should be carefully explored, particularly if otherwise unexplainable pain or evidence of intestinal obstruction are present. A discussion of the various deformities and joint diseases does not come within the scope of this book.

Syphilis.

Periostitis.

Rachitis and scurvy.

Congestion, edema and cyanosis.

Bubo.

Hernial openings.

THE BACK.—The deformities of the spine are essentially four in number. (1). *Lateral curvature* (scoliosis) most frequent in young girls and due to faulty position in standing and sitting, to muscular weakness or to general debility as from rachitis. It also results from chronic disease of the lung or pleura, paralyses affecting posture and mollities ossium. The deformity is seldom extreme in its ordinary form and is usually remediable. (2). *Lordosis* or exaggerated normal curve as exemplified in the later stages of pregnancy. It suggests that condition, abdominal tumors, ascites and pseudo-muscular hypertrophy. (3). *Kyphosis*, if sharply angled posterior projection of the spine is present Pott's disease (spinal caries) or mollities ossium are suggested. It is common however as a simple backward projection in the dorsal region, in rickets, debility and chronic emphysema. *Immobility of the spine:*—Temporary stiffness may be due to muscular strain or rheumatism, and chronic rigidity indicates arthritis deformans, paralysis agitans, Pott's disease or the curious *spondylitis rhyzomelique*, a painless disease of middle aged men, commencing in the hip joints and extending upwards to the spine and shoulder, the result being a rigid kyphotic spine and anchylosis of other affected joints. In other cases the disease originates in and is limited to the spine. *Tumors and Swellings.* A great variety are discussed in surgical and orthopedic works and the congenital spina bifida abscesses due to caries, fatty tumors, bed sores and sacral edema are frequently encountered.

Deformities.

Wide variation in significance.

THE JOINTS.—The following points should be determined:—(a). *Presence, extent and character of redness, swelling and deformity.* (b). *The position assumed.* (c). *Heat.* (d). *Tenderness.* (e). *Fluctuation or edema.* (f). *Degree of movement in relation to pain.* (g). *Presence of crepitus.* (h). *Outline of bony structures.* (i). *Anchylosis.* (j). *Atrophy.* (k). *Contractures.* (l). *Associated or antecedent injury, suppuration, excessive exhaustion, fatigue or other recognized causative or predisposing factors.* In the knee joint, fluid is most readily detected by fully extending and supporting the extremity and making downward pressure upon the patella which may be felt to tap the articular surface (floating of the patella). Ordinarily the outline of the distended synovial membrane in such a case is characteristic.

Floating patella.

General Comment.—It should be remembered that in *acute rheumatism* the larger joints are chiefly and primarily affected, usually bilaterally and in a definite order, and that the skin is moist, the sweat acid, fever and anæmia marked. In *acute gout* a single joint, usually

Large joints chiefly.

Chiefly small joints.

the great toe is primarily affected, a preference is shown for the small joints, the actual pain and throbbing are greater and more paroxysmal than in rheumatism in which they are chiefly excited by move-

Barometric influence.

ment though of the slightest degree. In *chronic rheumatism* deformity is as a rule not extreme, fever is absent, changes of the weather markedly affect it and exacerbation and subsidence are the rule. In *arthritis deformans* an acute primary attack cannot at first be distinguished from acute rheumatism, but it is more persistent, yields less readily

Resists salicylates.

to specific treatment (salicylates, etc.), and tends ultimately to become chronic, the joints grating on movement, deforming and becoming fixed. Both large and small joints are involved and the disease may become almost universal.

Septic Arthritis.—This occurs chiefly in the course of or immediately

Secondary to acute infection.

following acute infectious diseases, especially scarlet fever and dengue, and is common in pyæmia and gonorrhœa. The infantile form of septic arthritis may occur without an assignable cause and progress rapidly to suppuration. The various *tuberculous lesions* are too numerous to permit of discussion in this volume. One of the commonest and most regrettable of medical blunders is involved in the misdiagnosis

A common error.

of osteo-myelitis too frequently diagnosed as rheumatism. The urgent need of radical surgical measures in these cases makes it important that one should remember that it is the epiphyses that are chiefly involved rather than the joint itself and that the disease is distinctly septic in type, pain extreme, deep seated and boring, and further that as in rheumatism, several joints may be involved. So also in the hem-

Another source of error.

orrhagic diathesis such as hemophilia, scurvy and purpura joint, affections occur either rheumatic in character or *due to hemorrhage.*

In connection with organic nervous disease we meet with arthropathies. In *locomotor ataxia* and *syringomeylia,* one of the large joints,

Charcot joints.

most frequently the knee, ankle or hip may become suddenly swollen with or without pain and go on to rapid disintegration (Charcot joints). In connection with all chronic joint affections there may be marked muscular atrophy but *the electrical reactions are preserved. Hysterical*

Hysteria and chorea.

joints may create much confusion being excessively tender and frequently contractured. Chorea is frequently associated with what is probably a true rheumatic arthritis and myelitis may be accompanied by the same condition.

VOICE AND SPEECH.—One should train himself to carefully note variations from the normal healthy tone and enunciation. Even

Phthisis.

the laity recognizes the curiously soft high-pitched voice of the phthisical

patient, often indeed encountered in those showing merely a tuberculous predisposition; so also one recognizes the harsh voice of the prostitute and the thick muffled tone of the victim of tonsillitis, adenoid disease, or otherwise obstructed nasal passages. *Hoarseness* and *aphonia* are at times symptoms of great importance, pointing to acute or chronic laryngeal diseases; the *whispering voice* may be due to tuberculosis or malignant laryngitis, hysteric aphonia or paralysis of the abductors, due oftentimes to the pressure of an aneurism or mediastinal growth upon a recurrent laryngeal nerve. *Slow, scanning, syllabic speech* combined with an intention tremor suggests disseminated sclerosis or very rarely Friedreich's ataxia. In paralysis agitans the *crescendo speech* may be encountered; in this a sentence is begun slowly and with hesitation, but increases in rapidity and ends in a storm of words. In glossolabio-pharyngeal paralysis, amyotrophic lateral sclerosis and pseudo-bulbar paralysis, one encounters a mumbling speech, associated with tremor and atrophy of the tongue and curious immobility of the lips.

The prostitute.

Nose and throat.

Hoarseness and aphonia.

Syllabic speech.

Crescendo speech.

Mumbling speech.

APHASIA.—For normal processes it is necessary that the centres and mechanism for the perception and recollection of spoken and written or printed words, of articulation and of association, should be normally active and harmoniously interacting. It is evident that various disturbances may exist according to the character and location of the lesion. Furthermore, that we must discriminate between aphasia and the cases of paralysis or palsies of the muscles of articulation (anarthria). True aphasia is usually of central origin, the peripheral mechanism being unaffected.

Anarthria vs. Aphasia, aphemia and amimia.

Motor Aphasia.—This must be recognized as distinct from sensory aphasia, it is characterized by inability to voluntarily express by speech (aphemia), by writing (agraphia) or by gestures (amimia) what may be a perfectly clear mental image.

Sensory Aphasia, on the other hand, indicates a failure to understand or recognize spoken words (auditory aphasia, word deafness) or written words (visual aphasia, word blindness). A large number of subdivisions occur under both types, such as the loss of power to carry a tune or write music, or on the sensory side a failure to interpret or recognize musical sounds or notation. Still another variety (paraphasia) occurs in which wrong words or characters are spoken or written, or the same word repeated.

Word deafness and blindness.

Paraphasia

Apraxia.—By this is meant a loss of the faculty of recognizing or appreciating the identity, nature and uses of objects, and here again one has various sub-divisions, such as mind blindness (visual amnesia),

mind deafness (auditory amnesia), as well as terms indicating an inability to recognize and interpret odors, tastes, etc.

Purposive movements.

TREMOR.—Modes of Testing Tremor.—To distinguish between *passive tremor and intention tremor*, direct the patient to make some movement such as is involved in taking up and fastening a collar button, buttoning the vest, or drinking a glass of water. In this latter variety the tremor is greatly increased by the co-ordinate movements involved, and indeed may be wholly absent when the patient is at rest.

Extension increases them.

All tremors of the extremities are increased by extension, therefore the patient should extend the arm or leg in front of him, keeping the fingers or toes separated as widely as possible.

Facial tremor.

Again if the tips of the fingers are allowed to rest upon the physician's palm a vibration otherwise imperceptible may be readily detected. Tremors of the muscles of the face may be emphasized by the firm closure of the eyes, elevation of the eyebrows, or by drawing the corners of the mouth down or outward and tremor of the tongue is markedly increased by protrusion.

Tongue.

Action vs. Repose.

Fine and coarse.

In the case of individual muscles or muscle groups in various portions of the body, putting these in action or under continued strain usually increases their movement. One should note whether he is dealing with a *local or general, passive or intention, course or fine, rapid or slow, regular or irregular tremor.*

Trivial tremor.

Conditions with which Tremor is Associated.—The tremor of advancing age and the extremely rare condition of congenital or inherited tremor are unimportant, as are those due to temporary nervousness or overstrain and the excessive use of tea, coffee or tobacco.

In acute disease.

Tremor in acute disease usually indicates a profound toxæmia, being one of the unfavorable symptoms, for example, in typhoid fever, and it is often encountered in the typhoid state whatever be the underlying original disease.

Alcoholism.

In alcoholism it is not only a prominent feature of delirium tremens, but may often enable the physician to detect a recent debauch or present excessive potations, and if marked suggests the onset of an acute attack. Such a tremor is ordinarily fine, regular and persistent, and very similar to that of drug habituation.

Rapid and fine.

Exophthalmic Goitre.—In this disease the tremor is fine and rapid and if associated with a large thyroid and rapid pulse with or without exophthalmos, the diagnosis is made. Every suspected case should be tested for tremor by taking the finger tips in the palm of the hand.

"Pill-rolling."

Paralysis Agitans.—This is ordinarily a relatively slow tremor, first affecting the thumb and forefinger and causing a rolling movement of the thumb over the forefinger known as the "pill-making" or "bread-

crumbling" tremor. It disappears during sleep and *unlike intention tremor is checked by volitional movement.* In its more advanced form it may involve the arms, legs and head.

Mineral Poisoning.—Any case of otherwise unexplained tremor should suggest the possibility of poisoning by some metal or drug, such as cocaine, opium, arsenic, lead or mercury.

Disseminated sclerosis.

Intention Tremor.—This condition suggests always disseminated sclerosis, but may be met with in a marked form as a senile tremor or even in lead poisoning.

Fibrillary flickerings.

Tremor of the Muscles of the Face.—Twitching or flickering of the facial muscles strongly suggest profound neurasthenia, paresis or chronic alcoholism. In progressive muscular atrophy juvenile or spinal (adults) it may be observed in other regions as well.

Gastrocnemius cramp.

SPASM, CRAMPS, AND CONVULSIVE SEIZURES.—The term cramp should be reserved for a *painful*, spasmodic, muscular contraction temporary and localized. It may be associated with alcoholism, gout, diabetes, Bright's disease, hysteria, or excessive muscular fatigue and most commonly involves the calf muscles. The constant over use of one set of muscles such as is met with chiefly in the occupational neuroses as typified by the so-called scrivener's palsy may take the form of cramp, though frequently a simple spasm, mere weakness or an actual paralysis. (see p. 42). The term is quite correctly applied to painful spasm of the viscera such as is involved in renal or biliary colic.

Occupational cramps.

Cramp colics.

Causes.

Wry Neck.—This may be congenital, traumatic or purely spasmodic and save for cases associated with actual traumatism at or subsequent to birth, represents a disturbance affecting the spinal accessory nerve. In true congenital wry neck there is atrophy of the sterno-mastoid (usually the right) and facial asymmetry, as opposed to mere muscle callous due to rupture at the time of delivery. Spasmodic wry neck may be tonic, clonic, or more rarely combined, and may follow injury or exposure or be a true neurosis. In any event the muscle group of spinal accessory innervation is chiefly involved and the spasm of the rotators elevates the chin and swings it towards the unaffected side. The shoulder may be raised and the head drawn decidedly backward if the trapezius be much involved. Bilateral spasm drawing the head back and the face upward is rare. The clonic spasms are particularly distressing and unfortunately the disease usually tends to become fixed.

Varieties.

Trapezius involvement.

Bilateral wry neck.

Spasmus nutans.

Nodding Spasm.—This condition is usually a neurosis particularly in its adult form. In children it may be mimetic, reflex (teething), or associated with rickets. Ocular spasm especially nystagmus and less

often strabismus may be associated with it. The oscillations cease during sleep and are increased by excitement or when under observation and associated mental defects are occasionally noted. The reflex cases may be purely transient.

Tetany.—This condition is entirely distinct from tetanus, being merely a bilateral tonic spasm of the extremities, either continuous or paroxysmal, most commonly involving the feet and hands, less often the face, neck and jaw (trismus), rarely affecting the muscles of the trunk. Excessive muscular irritability is evidenced by the active contraction following compression of the brachial artery (Trousseau's sign) or even the lightest tap. Paresthesia may follow the lightest presure upon a sensory nerve, fever is sometimes present, and the disease is usually paroxysmal in type and associated with marked debility. Among the rare causes are gastric lavage, (in cases of dilatation), and removal of the thyroid gland. The disease is sometimes epidemic but is extremely rare in this country.

Parts involved.

Trousseau's sign.

Paramyoclonus Multiplex.—This syndrome covers those peculiar cases occurring chiefly in males and of unknown causation, in which clonic with occasional tonic spasm occurs, either constantly or paroxysmally, unaccompanied by sensory disturbances. These are usually symmetrical and rhythmical and cease during sleep.

Athetoid Movements.—These curiously deliberate, writhing, twisting movements of the fingers and hands, more rarely of the feet and toes, may result from infantile palsy or follow hemiplegia in adults. They are slower and less jerky than the movements of chorea with which they are sometimes confounded.

Myoidema.—One frequently notes a marked fleeting, localized contraction if a muscle be sharply tapped with the finger. This phenomenon is especially common in the muscles of the chest in connection with tuberculosis, but is not a sign of marked importance.

Catalepsy.—This extraordinary symptom indicates hysteria, but may be encountered in melancholia, brain tumor, meningitis, or be produced by hypnosis of which it constitutes the secondary stage. The patient appears asleep and the limbs remain for minutes or hours in the position in which they are placed. It is often auto-hypnotism, the eyes are closed, rolled upward, the pupils contracted and sensibility to pain and special and general sensation is abolished. The duration of such attacks is often short, but in the cataleptic sleeping girls (of the newspapers) the attacks may endure for years, such cases being able to hear, take occasional nourishment, and respond to other stimuli.

Auto-hypnotism.

Hysteria.—The actual study of cases is necessary to an understanding of the physiognomy of external appearances of this extraordinary condition, but once a large number have been carefully observed, the difficulties are largely done away with. The topic has been dealt with entire and the reader is referred to page 615.

INFANTILE CONVULSIONS.—Attacks precisely similar to those of epilepsy or differing in some particulars may be observed in both children and adults. In the former, they may be due to general malnutrition, either primary or secondary to disease, to rickets, to fever as is so frequently seen at the onset of acute infectious diseases, rarely in congestion of the brain and quite commonly as reflex phenomenon due to peripheral causes. Disease or irritation such as dentition with its associated digestive disturbances, phimosis, diseases of the eye and ear, and possibly intestinal worms may be causative. Convulsions accompany the larger number of cases of cerebral disease in children and one observes occasionally in young infants a gradually diminishing tendency to convulsive seizures from the time of their birth.

Simulate epilepsy.

Malnutrition, toxæmia, reflex causes.

Symptoms.—Aside from the usual premonitory symptoms such as slight switching, teeth grinding, restlessness or irritability the attack is precisely similar to that of epilepsy. The diagnosis in repeated attacks most often depends upon their subsidence or disappearance, when a cause is found and removed. The infant mortality from this source is considerable and unfortunately a large number of true epilepsies commence during the first three years of life, over a third of those investigated by Osler occurring in the first year.

Mortality.

Age.

Diagnosis.—In any case the first thought should be of the onset of *an acute infectious disease*, or *simple overloading of the stomach* and indigestion, two causes which account for by far the greater number of such attacks. *If the condition is persistent* we have not only to think of epilepsy but of the various diseases affecting the brain and its coverings. Adenoid disease should always receive consideration.

Seek a cause.

STATION. ATTITUDE. GAIT.—Static ataxia is tested by having a patient stand with heels and toes together and eyes closed. Marked swaying (more than an inch or so of excursion) suggests locomotor ataxia, Ménière's disease or a lesion of the mid cerebellar lobe. The patient may sway violently and fall unless closely watched.*

Test for static ataxia.

DECUBITUS.—In the bedfast patient, its chief divisions are the *active*

* One sufferer aptly described his sensation as that of a "chicken on a clothes line."

and passive, dorsal and lateral and it varies with the condition of the patient and the localized nature of his disease. Even the severer types of *pneumonia* do not rob the patient of his active decubitus, which contrasts strongly with the passive decubitus typified by severe *typhoid fever* in which the patient lies relaxed and helpless, must be moved from side to side, and occasionally straightened out to relieve the cramped posture or prevent his gravitating to the foot of the bed. The terms *dorsal* and *lateral* explain themselves but it is important to note whether the patient changes voluntarily from one to another or whether through extreme weakness, or because movement to a changed position involves increased pain, he maintains a fixed posture. In general the man with *appendicitis* lies on the back, often with the right knee drawn up, or in *general peritonitis* with both knees so placed. In severe abdominal colics (hepatic, renal, etc.) the knees are often drawn close to the belly and the *lateral coiled position* is common. In the early stage of a *pleurisy*, with or without pneumonia, the patient usually lies upon the affected side, probably to limit movement of the affected lung and facilitate that of the sound one, later in the disease the position is more variable. An *acute meningitis* is characterized by rigidity and retraction of the head and neck giving it a distinctive appearance. In a severe *rheumatic fever* and certain cases of *infantile scurvy* and *rickets* the patient lies rigidly in a dorsal position, his whole attitude expressing apprehension when his bed is approached or any effort made to examine or handle him. In cases of heart disease in which compensation has failed through whatever cause, and in asthma and severe emphysema or laryngeal obstruction, one is likely to find the peculiar sitting posture indicative of orthopnea. *In the terminal stage of mitral lesions* there is often a peculiar listless rolling of the head from side to side which with the anasarca, blurred features, cyanosis and orthopnea makes a characteristic picture. In some cases of *aneurism*, *acute aortitis* or *malignant* growth involving the mediastinum,* the patient not only sits up but must lean forward so as to rest the head upon the knees or upon some special support provided. In a case of Hodgkin's disease coming under the author's observation this attitude was assumed for weeks before the patient's death. So also in diaphragmatic pleurisy the patient sometimes sits inclined towards the affected side. Most careful attention should be given to the attitude of an unconscious patient with particular reference to paralysis, often-

Active vs. Passive.

Transient or fixed?

Appendicitis and peritonitis.

Pleurisy.

Meningitis.

Scorbutus and rachitis.

Asthma and heart disease.

Aneurism.

Mediastinitis aortitis.

Diaphragmatic pleurisy.

Paralysis.

*The same attitude may be assumed occasionally in extreme incompensation and asthmatic seizure.

times the appearance and passivity of an affected member or side is characteristic.

GAIT.—When practicable the legs should be uncovered from the hips down and in women the nightdress or chemise may be brought forward between the legs and fastened above so as to permit free observation and movement. *The patient should walk briskly and then slowly, with the eyes open and shut, stop and turn sharply at command, and follow a rug border or crack* at right angles to the line of vision. Due allowance must be made for nervousness and careful watch be kept lest a serious fall occur in some ataxic patient. The ordinary *limping gait* is to be referred to its proper cause, i.e., acute or chronic joint disease, flat foot, corns, etc. *A tilting forward* of the body is noticeable in paralysis agitans, kyphosis due to vertebral caries, and certain painful abdominal affections both acute and chronic. *Leaning backward* or an actual lordosis may be due to spinal disease, advanced pregnancy, abdominal tumors, ascites or obesity and is an important symptom of pseudo-hypertrophic paralysis and cretinism.

Necessary manœuvres.

Limping.

Paralysis agitans.

Leaning backward.

The gait of the ataxic stage of locomotor ataxia is striking, the feet being raised suddenly, jerked uncertainly forward beyond the ordinary limit and brought down heel first with a stamp. The patient keeps them well apart and must keep his eyes upon the ground, lest he sway or even fall. A sudden turning movement or an abrupt rise from the sitting posture is to him difficult or impossible. It must not be forgotton that a patient may have the disease for years before this *typical* gait appears, but even in such cases the acute observer may detect some of the peculiar features. In marked contrast is the *spastic gait* characteristic of *hemiplegia if unilateral, of lateral sclerosis if bilateral.* The rigid leg moves stiffly, the foot does not quite clear the ground, necessitating a tilting of the pelvis at each step, the toes describing an arc ("mowing gait") and wearing down the sole on its inner side. *Spastic contraction* of the adductors sometimes produces a *"cross legged progression"* also seen in hysteria. The *steppage gait* is most often seen in multiple neuritis as a result of foot drop, because of which the patient must lift the foot to raise the toe clear of the ground and the result is a paw-ing movement, the patient appearing to step over constantly recurring though non-existent obstacles.

Tabes dorsalis.

Mowing gait.

Cross-legged.

Prancing gait.

The Choreic Gait.—When present is that of the school boy, who in the schoolroom intentionally trips over his heel to attract the attention of his fellows. It is seldom unassociated with other characteristic symptoms. The *reeling gait* of cerebellar (vermis) lesions differs in no

Titubating gait.

Waddling gait.

respect from that of a drunken man. The *waddling gait* of pseudo-hypertrophic paralysis is due to the weakness of the glutei muscles. The pelvis and head of the femur are jerked forward at each step, the flexed knee is advanced, extended only after the foot is placed flat upon the ground, and the patient cannot stand on tiptoe. The *festinant* gait is pathognomonic of paralysis agitans and the patient walking as if trying to recover from a thrust from behind, trotting or shuffling with the body bent forward.

Festinant gait.

Intermittent claudication and Thomsen's disease merit special reference. The former is associated with arterio-sclerosis and deficient circulation in the lower limbs, characterized by numbness or *painful cramps* on standing and walking, associated with rigidity, paræsthesia or disability, often entirely absent when at rest. The latter, a family disease, is characterized by stiffness or *painless contraction* on attempting any muscular action. This in walking at first checks or delays each step but gradually wears away and permits normal progression, only to re-appear when any new muscle group is called into action or the same action repeated after a rest. *In the former disease the pulse is usually absent below the knee during the seizure.*

Erb's dysbasia.

Myotonia.

Astasia.—(Inability to stand). **Abasia.**—(Inability to walk). This must be classed as a functional neurosis inasmuch as all other nervous functions are normal and the picture strongly suggests hysteria. Walking may be impossible yet all its *movements* may be normally performed when the patient is lying in bed. The inability may be absolute or partial, giving rise to what simulates spastic paralysis on the one hand or flaccidity upon the other. *Saltatory spasm* is sometimes observed.

A bastard syndrome.

Hysteric or neurasthenic.

WEIGHT AND HEIGHT.—Every doctor's office should contain a pair of accurate scales, and a knowledge of the weight, *past and present*, is of the utmost importance both in diagnosis, prognosis and treatment. In general one encounters a tendency to increase in weight after the age of 40 and *corpulence is ordinarily associated with a weak musculature, poor resistance to acute disease and a decided tendency to degeneration of the heart, blood vessels and kidneys in middle age. Early loss of weight* is one of the most significant signs of incipient tuberculosis and carcinoma of the stomach as well as many other chronic ailments. *Wasting or emaciation* is pronounced in certain digestive disorders, all severe or prolonged fevers, true diabetes mellitus and the various wasting diseases of children. *A very fair estimate of weight loss can be made by pinching up a fold of the skin over the upper arm and noting its looseness.* In chronic exhausting diseases not only is

Obesity.

Early weight loss.

Wasting.

Lax skin.

the skin a misfit but it lacks normal elasticity. *Increase of weight* ordinarily indicates an improved general condition and the arrest of any local disorder, but it must be remembered that *edema also increases it*, and in Bright's disease especially, the variation in body weight may reveal a hydremia not shown by outward signs. So also loss of weight may in this and in cardiac types of edema be a most favorable symptom. Weight loss always accompanies profuse diuresis, sweating or purging. *Children should be weighed weekly* and should show an increase of from 20–25 grams each month for the first four, from 12–20 grams for the five succeeding and somewhat less for the remaining months of the first year of life. *During the first days after birth they may show a considerable weight loss.* Weighing should be done at the same hour in relation to meals in all cases and with the same amount of clothing if possible inasmuch as the estimate of five to seven pounds (for clothed adults) usually made is subject to grave error. *The relation of height to weight* is important and is well shown in the table which appears upon the opposite page. This was prepared by Dr. O. H. Rogers and is ingenious and accurate. Insurance companies regard with suspicion men whose weight runs more than 20 or 25% above the standard as indicated by the table and that this prejudice is well founded has been shown by the results of the recent combined actuarial investigation which has proven a high mortality in this class of lives. The table of

Edema.

Infants.

Precautions.

Height and weight.

Life insurance.

FOR AGE FORTY-SEVEN AND UPWARD.

Height.	Normal Weight.	—20 Per Cent.	+20 Per Cent.	+30 Per Cent.
5 feet.....	134 pounds.	107 pounds.	161 pounds.	174 pounds.
5 " 1 inch.....	136 "	109 "	163 "	177 "
5 " 2 inches...	138 "	110 "	166 "	179 "
5 " 3 " ...	141 "	113 "	169 "	183 "
5 " 4 " ...	144 "	115 "	173 "	187 "
5 " 5 " ...	148 "	118 "	178 "	192 "
5 " 6 " ...	152 "	122 "	182 "	198 "
5 " 7 " ...	157 "	126 "	188 "	204 "
5 " 8 " ...	162 "	130 "	194 "	211 "
5 " 9 " ...	167 "	134 "	200 "	217 "
5 " 10 " ...	172 "	138 "	206 "	224 "
5 " 11 " ...	178 "	142 "	214 "	231 "
6 "	183 "	146 "	220 "	238 "
6 " 1 inch.....	188 "	150 "	226 "	244 "
6 " 2 inches...	194 "	155 "	233 "	252 "
6 " 3 " ...	200 "	160 "	240 "	260 "

For younger ages subtract ½ pound for each year under forty-seven, and the result will be the normal weight for the given age.

D. H. Wells is also given and is an excellent one for practical use. In connection with *underweights* a family history of tuberculosis or personal history of past infection, a narrow chest, small heart and the evidence of poor circulation are extremely important. So also in *overweight* one must distinguish between those who have big bones, firm well developed muscles and moderate abdominal girth as compared with the flabby, big bellied sedentary livers and heavy eaters, particularly if these have a family history indicating an hereditary tendency to apoplexy and diseases of the heart and kidneys. *Very tall, slender men, giants and professional athletes are not as a class long lived.*

Underweight, significance.

Overweight.

AGE—SEX—HABITS—SOCIAL STATE AND RESIDENCE.

Age estimation.

AGE.—By practice and close observation one can usually estimate closely the age of patients, though like the census enumerator he may at times in the case of women be obliged to accept statements with a mental reservation. The essential points are *apparent* as compared with *actual* age and *unduly rapid aging*. Certain ages represent a special predisposition to certain diseases, acute and chronic. *In infancy and childhood* we meet especially with acute digestive disturbances, rickets, the exanthemata, affections of the lymph glands, meningitis, infantile palsies, chorea, croup and cretinism. Tuberculosis attacks the young with an especial frequency and a higher mortality, but in those under the age of puberty it is peculiarly liable to affect the lymph glands, particularly those of the cervical triangles or abdomen. *During adolescence*, chlorosis, various forms of hysteria, epilepsy, acute rheumatism, gastric ulcer and tuberculosis are extremely common. *As middle age approaches* the tendency to the exanthemata and to acute infections generally is diminished and a predisposition to degenerative diseases of a chronic type appears. Such are arterio-sclerosis, myocarditis, valvular diseases, gout, gall stones, diabetes, the insanities, various forms of paralysis, profound blood disturbances such as leukæmia and pernicious anæmia, carcinoma, chronic Bright's disease and asthma. *In old age*, this predisposition of middle age is, in certain conditions, intensified and in addition we find prostatic disease, chronic bronchitis and emphysema, and a return to the childish tendency to broncho-pneumonia. In some instances diseases are wholly limited to certain age periods, but usually in weighing diagnostic probabilities the question is one of *relative* frequency. For example, cancer of the stomach is as rare in a person under 20 as is mumps in one over 70. Rapid aging may bring the man of 30 to the same status as the one of 70, and on the

Apparent vs. Actual.

Diseases of infancy and youth.

Middle age.

Old age.

Rapid aging.

other hand, the latter, by reason of inherited vigor of constitution and right living may be physically 20 years under his actual age. Every clinician repeatedly encounters cases of marked arterio-sclerosis in men under 30 years of age. The inheritance of poor structural material, dissipation, and mental strain are the chief factors in early aging. Finally, one must remember *the effect of age upon prognosis.* The acute exanthemata though more readily acquired are for the most part much more lightly borne by the child than by the adult and resistance to tuberculosis is much more marked in persons above the age of 30. Pneumonias are peculiarly fatal at the extremes of life and the *young old* people strongly resist the advance of chronic disease.

Young old, and old young men.

Age affects prognosis.

SEX.—In general, men and women suffer about equally and resist in about the same measure the larger number of diseases, nevertheless there are certain striking differences both as to incidence and severity. As compared with men, women, through the disabilities peculiar to their sex, appear to develop superior endurance particularly marked in chronic disease. The woman is especially liable to neurasthenia, hysteria, movable kidney, myxœdema, arthritis deformans, chlorosis and chronic secondary anæmias, gall stones, goitre, cancer of the breast, and gastric ulcer. On the other hand, she almost escapes hæmophilia, aneurism and locomotor ataxia, and suffers less often than the man from acute infections, gout, appendicitis, typhoid fever, certain diseases of the heart, progressive muscular atrophy, diabetes, carcinoma of the stomach and bowel, leukæmia, pernicious anæmia and Addison's disease.

Predisposition.

Immunity.

Race.—The special predisposition of the Hebrew to diabetes mellitus, of the Irish to tuberculosis, of the English to gout, of the continental races to suicide, etc., etc., is well known. Moreover certain races show markedly greater resistance to disease and surgical procedures than do others.

HABITS AND ENVIRONMENT.—The term "habits" should cover a much larger field than the patient's indulgence in tobacco, drugs, or alcoholics and include his environment and mode of life, his hours for meals and for sleep, his manner of eating and the nature of his meals, no less than the extent of his drinking. One should ask specifically what was taken for breakfast or lunch preceding the time of the examination, or just what is usually taken at such a time, how much water is drunk and when, how much time is taken for the meal, whether the food is properly masticated, and indeed ascertain the condition of the teeth by direct inspection when examining the tongue and

Scope.

Specific inquiry.

Teeth.

throat. The author believes that too much stress is laid upon the use of tea, coffee and tobacco, and too little upon the idiosyncrasy of the patient in regard to these articles. That which constitutes excess for one represents moderation in another and the slight amount representing the average consumption can ordinarily be of little consequence; hence it is a positive hardship to arbitrarily shut off rather than to sensibly cut down the tea, coffee or tobacco which for years has been the solace of some elderly patient. As regards *tobacco*, especially, one may say that the signs of *overindulgence* are found in unrefreshing sleep, furred tongue and bad taste in the mouth, nervousness which takes the form of mental irritation and perhaps tremor, and at times a distinct disturbance of digestion and irritability or palpitation of the heart. In the man under middle age these symptoms mean little; in the elderly man much, and for the latter, persistence in excess is fraught with danger.

Tea, coffee and tobacco.

Its dangers.

Misuse of terms.

The Use of Alcohol.—The common mistake of the novice is a failure to bring out tactfully yet fully the facts bearing upon a patient's alcoholic indulgence. He is too easily satisfied when the patient says he "does not drink at all," or he "takes an occasional drink," or "not enough to hurt him," "a drop now and then," "just a social glass," etc., etc. An absolute denial of overindulgence is usually proudly made by the man who has been a hard drinker for years but has stopped, it may be only for a few days. The "now and then" kind, or those who say "often not for a year," frequently represent the worse type of spree drinkers. One should inquire therefore about the present and the past, the extent, the hours, the kind, in what relation to meals and with what affect upon the health. It is impossible to lay down an absolute rule as to what constitutes excess and it is probable that the man who takes a little liquor, even two or three times a day well diluted and on a full stomach, may suffer little or no bad effects, but the American style of drinking is peculiarly harmful, as taking the form of "cocktails" before meals, and a pernicious multiplicity engendered by the custom of "treating." The three diseases most often due to such overindulgence are:—delirium tremens, alcoholic neuritis, and cirrhosis of the liver, but it is a powerful contributory factor in an enormous number of chronic diseases, inviting and promoting their development and shortening life and in severe acute diseases such as pneumonia the drunkard stands little show.

Excess.

Diseases induced.

Resistance to disease.

Opium and cocaine.

Drug Habit.—As regards the use of drugs, we have chiefly to deal with the various forms of opium and with cocaine. Neither produces in every individual a clear and definite syndrome but one of the pri-

mary indications is a peculiar nervous instability, or a baffling and bastard symptom complex. A markedly dilated pupil in cocaine users or a markedly contracted one in the victim of the opium habit may attract attention, but in either at the time of examination the pupil may be normal. If one examines such patients thoroughly, he will often find recent hypodermic punctures, black dots representing old ones or evidence of recent or old multiple abscesses due to the use of dirty needles. Such are usually on the right thigh or left arm of a right handed person, and the needle is usually introduced directly through the clothing. The peculiar pallor sometimes present has been referred to, the skin is likely to be dry in morphine users, the appetite poor and bowels constipated and the victims of either drug are subject to fits of profound depression succeeded by periods of buoyancy. "Nose rubbing" may suggest opium and in the case of cocaine users especially formication is not an infrequent symptom. The excessive use of cocaine may be honestly denied by one who has contracted the habit through the use of the drug in the nasal passage or throat, and the author has encountered many such cases, the patient not realizing that absorption takes place in such instances as readily as if the drug were actually taken into the stomach. The overuse of such drugs as acetanilid may become a fixed habit and lead to serious or even fatal illness.

Difficulties encountered.

Hypodermic marks.

Mental state.

Innocent victims.

Acetanilid.

MARRIED OR SINGLE.—The question of marriage is important chiefly in relation to the matter of pregnancy or childbearing in women and its after effects. Many obscure ailments in the female are traceable to unrecognized or neglected lacerations, while endometritis, uterine displacements, pelvic abscess and epithelioma are especially common in multiparæ. The number of pregnancies, the duration and severity of labor, miscarriages and the circumstances attending them, the number of children living and dead, the health of survivors and the causes of death of those deceased, are important. Marks of syphilis in a child should at once direct attention to one or both parents and vice versa, and tuberculosis and other diseases may be suggested in a similar manner.

Pregnancy and childbirth.

Miscarriages.

OCCUPATION.—The occupation and environment are important factors in diagnosis and prognosis and may throw light upon degenerative processes, functional and organic nervous disease and accidents. City dwellers and indoor workers suffer especially from dyspepsia, tuberculosis, neurasthenia and similar disorders. On the other hand, even the country dweller and out of door worker *may* suffer from the poor ventilation and deficient sanitation to be noted in many farm houses.

Sedentary vs. Active.

City vs. Country.

Exposure.

So also must one consider the effect of unusual exposure to cold, wet, and to such poisons as malaria. The best conditions are to be found in camp life or the homes of the better class of agriculturists, the *worst* in the slums, sweat shops or improperly ventilated factories of great cities. On the one hand there is the maximum of fresh air, sunshine and healthful exercise, on the other foul air in the shop and home, constant exercise of the same muscles and in cramped positions, and as a rule deficient or improper food. Certain occupations involving the continuous use of one neuro-muscular unit produce specific ailments as do those involving pressure, or irritation of a particular portion of the body. Cases of nystagmus have been reported as due to the cramped position of miners working with eyes fixed upon a particular point. Penman's cramp or scrivener's palsy is a familiar example, and the chronic laryngitis of military men, clergymen, auctioneers and public speakers falls under the same head. Another example is the inflammation of the patellar bursa, known as "housemaid's knee," the condition being encountered also in roof shinglers, tile layers and others working under similar conditions.

Fatigue neuroses.

Housemaid's knee.

School girls.

"Going to boarding school" is one of the worst of occupations if the school work is over-hard, the heating and ventilation insufficient, the food poorly prepared or lacking in quality and variety or the out of door exercise scant. Nothing breeds more chlorotics than a poor boarding school or makes healthier women than does a good one.

Barkeepers and the like.

Habits in Relation to Occupation.—The excessive use of intoxicants is definitely related to certain callings. The manager of a large hotel syndicate has said that he has yet to see one bar-keeper who failed to develop the drinking habit, and this statement undoubtedly expresses a rule subject to few exceptions. A traveling man who sells bar supplies, cigars, or even mineral waters is constantly subjected to a pressure which too often results in the formation of the liquor habit.

Combined factors.

Potency of nervous strain.

Occupation Involving Continuous Mental Strain.—In this day of great enterprises a large proportion of the chronic ailments encountered by the physician may be traced in whole or in part to continuous mental over-strain, usually combined with lack of exercise, improper diet and too often with over-indulgence in liquor or tobacco. No one who has seen the remarkable improvement in chronic disorders of the heart, stomach, kidneys, or various diseases of the nervous system, that follows a period of complete rest and freedom from worry, can doubt the potency of nervous strain as a factor in the etiology and prognosis

of disease. Railroad men, financiers and especially board of trade operators suffer greatly from this cause. The life of a practising physician is of such a nature as to readily explain the high early mortality encountered in this class, for in the physician's calling are combined deficient and interrupted sleep, lack of exercise and recreation, excessive and continuous mental strain, and a large demand upon the kindly emotions so constantly appealed to by his clients. The physician.

Mineral Poisoning.—A complete volume would be required to do justice to this aspect of our topic, and there can be no doubt that too little attention is paid to the possibility of occupational poisoning by physicians other than those practising in large manufacturing centres, where the conditions are exceptionally favorable to the development of occupational diseases.

Arsenic.—Aside from poisoning due to the inhalation of fumes or dust during the processes of milling, grinding and smelting one encounters cases amongst workers in aniline dyes, toy or artificial flower makers, dyers of woolen or cotton goods, playing card makers, taxidermists, lithographers and shot makers.

Aniline.—This derives its harmful effect from three sources viz.:—itself, the arsenic often combined with it, and the nitro-benzol used in its manufacture.

Bromine and Iodine.—The fumes of this substance cause bronchitis and predispose to tuberculosis.

Carbon Bisulphide.—This highly poisonous substance is used as a solvent for sulphur, iodine and oils and is largely used in the manufacture of rubber goods.

Chlorine.—In the bleaching of linen, cotton, bones, ivory and rags the fumes are irritating to the bronchial-mucous membranes and predispose to tuberculosis.

Chromium.—This enters into chrome yellow and chrome green, and is used in staining glass or porcelain, printing bank notes, and dyeing linen, wool and silk, and as potassium bichromate is used in photography.

Copper.—Workers in brass, nickel platers, bronzers, copper sheet scrapers, pin makers, bell metal workers, stone workers, engine wipers, and others handling copper, bronze, brass or nickel may suffer from chronic poisoning. It should be remembered that brass is composed of copper and zinc, with or without tin and lead, bronze, of all four metals, and that nickel plating is sometimes done with an alloy composed of copper, nickel, iron and tin. It is further combined oftentimes Workers affected. Composition.

with lead and arsenic. One of the most severe cases of lead poisoning ever encountered by the author was caused by polishing an alloy of this kind.

Often overlooked.

Lead.—The possibility of lead poisoning in obscure gastro-intestinal affections as in connection with its well known nervous manifestations should never be forgotten. Not only is it found in lead smelters, refiners, sheet-lead rollers, lead pipe makers, shot makers, type-setters, plumbers, toy makers, and painters, but also in lacquer polishers, gilders, bronzers, enamel workers, glaziers, all pot, pan, card, cardboard or brick makers, makers of brass instruments, file cutters, flint glass workers, workers in white or red lead and litharge, calico printers and those engaged in the manufacture of lace, artificial flowers and wall papers.

Suggestive occupations.

Mercury.—Chronic mercurial poisoning may result from idiosyncrasy, long continued over-dosage, or employment in smelters, quick silver mines and felting rooms.

Phosphorus.—This is now comparatively rare, and is almost wholly limited to those engaged in the manufacture of the parlor match.

Turpentine.—Some persons are peculiarly subject to turpentine poisoning, and painters working in poorly ventilated rooms may become chronically affected.

Occupations Involving Excessive Heat.—Extreme *dry* heat can be borne without serious results in persons habituated, nevertheless, firemen on ocean steamers or naval vessels, and bakers suffer from heat, moisture, foul air, and the effects of chill and exposure due to extreme temperature variations.

Miscellaneous Diseases of Occupation.—Millers, potters, file cutters, grinders of edged tools, wool and cotton spinners, marble and stone cutters are peculiarly susceptible to tuberculosis, particularly in its fibroid form. Handlers of rags and skins occasionally acquire anthrax, internal or external. Female domestics are peculiarly liable to anæmia, gastric ulcer and tuberculosis. Butchers or slaughter house men suffer from septic infections, stablemen from tetanus and glanders, brewers, saloon-keepers, bar-tenders and others of the same class show an enormous mortality from alcoholism, tuberculosis, diseases of the nervous system, pneumonia, diseases of the liver and kidneys, and suicide.

Tuberculosis.

Anthrax.

Sepsis, tetanus, glanders.

Suicide.

Occupational Stigmata.—Those who follow certain occupations often present somewhat characteristic deformities; the lowered shoulder of the desk worker, pack peddler and tailor are familiar to everyone. Few railroad trainmen escape for long without the loss of one or more fingers.

The lowered shoulder.

Crippled hands.

Callosities.

Furthermore, the study of the *callosities associated with certain callings* may at times prove helpful both in diagnosis and identification, and a few of the occupations thus suggested are briefly given below.

Banjo, Guitar and Harp Players.—Finger tips both hands. **Zither Player.**—Finger tips of left hand, under surface and tips of index, middle and ring finger of right. **Violincello or Violin Player.**—Tips of fingers, left hand only. **Compositors.**—Palmar surface of thumb and index finger, right hand. **Fencing Masters.**—Ulnar border, right palm. **Hand-organ Man.**—Outer side of right hip and thigh. **Seamstresses.**—Roughened radial border of terminal phalanges of left thumb and index. **Tailors.**—Ring shaped callous on right thumb and index finger, left thumb and index roughened, enlarged bursæ over external malleoli. **Turners.**—Outer border of right little finger. **Clerk Using Pen or Pencil.**—Outer surface of terminal joint, right little finger. **The Rough Callous Hand** of the day laborer contrasts sharply with the soft hand of the sedentary worker, as does that of the seamstress or domestic with those representing the lighter occupations of her sex. The tan of the sailor contrasts sharply with that of the soldier, the one wearing his shirt open at the front, the other the high collar.

Idleness a disease breeder.

General Comment.—Want of occupation is often deleterious and the neurasthenia and hysteria, morbid irritability and ennui of a certain class of women are rarely seen in the hardworking housewife. The man who for years has given his attention to active business or professional work may find complete idleness both boresome and dangerous and few men or women can be well mentally or physically without some definite occupation, and every man who in later years forsakes his life's work should cultivate a hobby or divert himself by travel.

Value of a "hobby."

Record important.

RESIDENCE.—The novice frequently fails to secure the full name and postal address of the patient under examination, forgetting that future communication is sometimes most important in that some of the most valuable information comes from the following up of unusual cases. *Both present and past residence should be known*, and, in general, information upon this subject is of value in relation to the possible introduction of epidemic disease by persons coming from an infected area, in tracing its distribution, as in an epidemic of cholera or typhoid affecting certain portions of a city or larger district, in tracing its individual sources as illustrated by the detection of careless dairymen, who spread infection along their route, and finally, because

Present and past.

Epidemics.

of the special liability to certain diseases in definite districts or countries, as illustrated by the diseases of the tropics, such as malarial fever, dengue, yellow fever, and plague, the hydatid disease of the Icelander, or the leprosy of the Sandwich Islander. Specific inquiry concerning the exact situation of the patient's house, its elevation, exposure, the character of the surrounding soil, and the location of the sleeping chamber, is often important.

The home.

FAMILY HISTORY.—*The transmission of special structural vulnerability and predisposition is just as marked and as well proven as inherited likeness in form or feature*, and in taking a family history, one should secure full information as to the terminal illnesses of family members, often going back two generations and including collaterals, and of the state of health or cause of death of the husband or wife of the patient, and the children. *It is seldom sufficient to ask whether any members of the family, immediate or remote, have suffered from hereditary disease, or even to put the question in the somewhat more specific form of an inquiry concerning tuberculosis or cancer.* Apoplexy, heart disease or Bright's disease may be readily admitted, but insanity, epilepsy, tuberculosis, and malignant disease are often concealed, either intentionally or innocently. The questions must bring out the *symptoms* of the illnesses of family members. One may ask if there has been chronic cough, spitting of blood, emaciation, night sweats or fever in a given case, or the vomiting of blood, or the presence of a tumor, associated with emaciation and failing strength. Deaths from "childbirth," "exhaustion," "grief," "broken heart," "general decline," etc., must never be accepted without careful and tactful cross examination. "Senility" or "old age" are much abused terms and in the lay mind may cover death at any age above 50. No man ever passed his novitiate as a case-taker, without saying hard things of "malaria," "chills," "fever," "decline," and especially "marasmus" and "don't know," and no student can give better evidence of his skill and tact than is furnished by a full and complete family history.

Inherited predisposition.

Specific inquiry.

Blind terms.

Alternatives in Heredity.—Few diseases are directly transmitted as such; rather does hereditary influence take the form of predisposition. Thus the disease of the descendant may be but the congener of that of the forbear and as such is termed an *alternative*, or, the inheritance may be that of marked vulnerability or its opposite, invulnerability. To the former class belongs the "short lived family," whose members show a low average life time and a ready acquirement of and feeble resistance to acute or chronic ailments. To the latter appertains a

Direct transmission rare.

Short vs. long-lived families.

vigor of constitution and strength and harmony of structure which ensures resistance to disease and long life.

Alternatives in nervous diseases.

The Nervous System in Heredity.—*A strong alternative relation exists between alcoholism, insanity, epilepsy, hysteria and criminal impulse.* In some families, however, there is little variation, insane grandchildren follow insane grandparents, epilepsy in the father means the same disease in the child, and some remarkable cases of hereditary alcoholism have come under the author's observation. In one especially it seemed impossible for any family member to escape the curse.

Cardio-vascular alternatives.

Apoplexy.—The hereditary tendency to cerebral hemorrhage is one of the most striking facts in medicine and is inseparable from its alternative conditions, arterio-sclerosis, chronic Bright's disease, aneurism and the degenerative diseases of the heart. The author has reported a case in which, of nine family members, five died of apoplexy, two of chronic Bright's disease, and one of heart disease, every death occurring between the ages of 40 and 55. The then surviving brother has since died of the dominant disease.

Distinctly hereditary.

Cancer.—Few physicians of experience will be found who deny the hereditary nature of cancer, yet it is well to avoid too radical statements in regard to it, as tending to create undue apprehension in the minds of members of tainted families. Like apoplexy the tendency in this disease is towards death in middle age, the transmission seeming to be more marked from mother to daughter than from father to son.

Race and heredity.

Diabetes.—Few diseases are more distinctly hereditary than diabetes, if we regard it as one of the alternatives of inheritance in nervous and mental diseases. According to recent observers it is possible to separate a distinctly hereditary form which is characterized by an equality in predisposition as between male and female, or even to predominance in the female. The best examples of inherited disease are found in the race showing the greatest vulnerability, namely, the Hebrew.*

Potent and protean.

Gout.—This distinctly hereditary ailment is potent in the production of arterio-sclerosis, apoplexy, Bright's disease, gastro-intestinal ailments and certain forms of diabetes, as well as all forms of gout.

Atavistic.

Hæmophilia.—This extraordinary disease is atavistic in its transmission, the deadly tendency to excessive bleeding, spontaneous or induced, being transmitted through the females of a family to the male issue. In certain families it has been traced back for hundreds of years in unbroken sequence.

Females transmit.

* The Hereditary Form of Diabetes. M. Loeb. Zentralblatt Für Innere Medizin, Aug. 12, 1905. No. 32, page 786.

Syphilis.—Direct inquiry is impossible and the physician must ordinarily depend upon the recognition of outward signs of inherited syphilis or in obscure cases apply the therapeutic test.

Direct transmission rare.

Tuberculosis.—That in rare instances tuberculosis may be transmitted from mother to child in utero cannot be doubted and it is certain that the incidence of tuberculosis is greater in those of tainted family record. Inheritance probably has to do with structural and constitutional conditions which render the child of tuberculous parents or antecedents more vulnerable and less resistant to the tubercle bacillus than the one of sound, vigorous and untainted stock. Even predisposed children, if favored in climate and environment, need seldom or never develop the disease, under conditions eliminating the possibility of contact with the living germ, the exception being found in a latent prenatal glandular infection. Unfortunately, however, these victims of predisposition born into tuberculous households, are in every way exposed, through their stature, their creeping, their tendency to carry dirty hands and infected articles to the mouth, and even through the embraces of an infected parent, brother or sister. The peculiar shape of the chest associated with tuberculosis and found most frequently in those of tainted stock is merely one of the structural peculiarities and disabilities which become factors in infection and in the course of the disease. Far more important are obstructions in the naso-pharyngeal passages, especially adenoid growths and hypertrophied tonsils. These, in themselves favorable soil for the tubercle bacillus, by their interference with free respiration prevent the development of the lungs and actually retract the chest while further contributing to infection through frequent recurring colds and chronic catarrh. One has only to consult insurance manuals to understand how important are physique and family taint as factors in tuberculosis, and it may further be said that admitting certain exceptions it is generally true that an exceptional physique largely overbalances a bad family history.

Diminished resistance.

Deadly home surroundings.

Naso-pharyngeal obstruction.

Life insurance.

Degree of relationship.

A source of error.

Special Conditions Affecting Heredity.—The significance of tuberculosis in the parents is *apparently* but slightly greater than that in brothers or sisters, but probably this is because cases of arrested tuberculosis in the parents are seldom to be traced in case-taking, such past events being usually either forgotten or concealed. Any one who has practised in a region frequented by cured consumptives will appreciate the truth of this statement. **Physical Resemblance.**—Mere facial resemblance is of little importance, but general structural likeness is distinctly so. **Exposure to Infection.**—The health of living family

members and especially of the children of parents under examination, as well as the condition of parents affected by or dying of tuberculosis *at the time of the birth* of any individual under examination, are important.

PREVIOUS ILLNESS.—A knowledge of the general state of health and past illnesses of a patient may assist the diagnosis, either by the exclusion of a disease of the permanent or temporary immunity-conferring type, or by fitting a symptom group to the known after effects of a previous illness. The exanthemata and yellow fever illustrate the one, syphilis both.

Immunity.

Misnamed Ailments.—A patient reporting "stomach trouble" may present a clear history of gastric ulcer, appendicitis or gall stones; another may have had repeated attacks of acute rheumatism or gout and our attention is thus directed to the heart, blood vessels, kidneys. A history of severe nocturnal headache may be explained by an ancient syphilis, or an intractable periodic neuralgia by past malaria. *In such interrogation the simplest and most homely phraseology should be used*, especially in dealing with the ignorant, and leading questions cannot wholly be avoided.*

Use plain language.

HISTORY OF "PRESENT AILMENT" AND ANALYSIS OF CERTAIN COMMON SYMPTOMS.

History of Present Ailment.—The patient should tell of his own volition but in as few words as possible the symptoms of his disease and their duration. Any attempt to theorize or drag in immaterial facts should be tactfully checked. To guide and direct the disclosures, check verbosity and triviality and yet use few leading questions is an art both valuable and rare. *One should discriminate between those symptoms that are general in their nature, common to a large number of diseases and subject to various interpretations from those that are local, peculiar or specific.* Some of the principal *general* symptoms will be described in this connection and only their special features referred to in other sections or under the symptomatology of specific ailments.

Check garrulity but limit leading questions.

FEVER.—Fever as a clinical manifestation is merely an elevation of the body temperature as formerly determined roughly by the hand

Thermometry.

* An ignorant patient of the author dying of previously unrecognized perforating duodenal ulcer readily admitted having formerly "puked" blood, though on repeated questioning he had denied "vomiting" of any kind.

but nowadays accurately by the clinical thermometer. In such diseases as pneumonia and typhoid the skin is dry and hot. On the other hand a high rectal temperature may exist in cholera and the surface temperature be as low as 70 degrees. The thermometer is ordinarily applied to one of four points, these being in the order of frequency, the *mouth*, the *axilla*, the *rectum* and the *vagina*. There is no poorer investment than a cheap clinical thermometer and the test scales supplied with some of those of the lower grades are worthless by reason of improper calibration, imperfect seasoning and misrepresentation on the part of the manufacturer. In the use of a thermometer, certain precautions must be observed:—

Selective points.

Worthless thermometers.

Preliminaries.

(a). *It should be cleaned before and after use.* (b). *The scale should invariably be inspected* as the thermometers are self registering and require to be shaken down after use. (c). If used in the mouth, *it should be placed well under the tongue and held by the tightly closed lips* (not teeth) *of the patient;* if, for any reason the lips cannot be closed, if the patient be unconscious or delirious or if an acute stomatitis or tonsillitis be present the temperature should be taken per rectum, or in the axilla. (d). *Axillary temperatures should be avoided wherever possible*, as being subject to greater error variation than in other regions. If so taken the thermometer should be placed deeply in the axilla which should have been freed from any moisture present and the elbow of that side should be close to the body and carried well forward. *Axillary readings are particularly misleading in incipient or but slightly febrile tuberculosis.*

Mouth temperatures.

Axillary readings.

Temperature Range in the Human Body.—Our physiologists state that in normal persons the range may represent 99.5° F. as a maximum, and 97.7° F. a minimum. *Sub-febrile* temperatures are those running from 99.5 to 101.3; *moderate fever*, 101.3°–103° F.; *high fever*, 103°–105° F.; *hyperpyrexia* 105°+. There is a normal daily variation reflected in nearly all fevers, the range being about 1° or 1.5° F., the lowest reading occurring in the early hours following midnight, the highest between 4 and 6 o'clock in the afternoon. Granting that 99° F. to 99.5 or even 99.8 may represent a normal variation in infants the author believes that the range as ordinarily given by the physiologists for the adult is too high and based upon observations which included persons having incipient disease, such as pulmonary tuberculosis. Years of observation in private and public practice have convinced

Normal.

Fever.

Faulty statistics.

NOTE.—To convert Fahrenheit readings into centigrade multiply the centigrade reading by 9, divide by 5 and add 32. (See table, p. 643.)

him that *under ordinary clinical conditions, a persistent maximum temperature exceeding 99°F. is strongly suggestive of incipient tuberculosis*, the onset of some acute ailment, or, at all events is referable to some pathological condition.*† By far the greater number prove to be cases of *incipient* tuberculosis, a condition recognized but rarely until within the last two decades. Incipient tuberculosis, etc.

It should always be remembered that violent exercise and excessive heat slightly and temporarily raise body temperature and that in infants and very old persons it may range half a degree or more higher than in those of intermediate age. The temperature of children moreover is easily affected and may reach a high degree without serious significance, often subsiding as suddenly as it came. For variable periods in certain cases of tuberculosis, acute or chronic, *the morning* temperature represents the maximum. Heat and exercise. Ephemeral fever. Inverse temperature.

Pathologic Variations in Temperature.—Hyperpyrexia of extraordinary degree has been reported, but has probably been due to deception on the part of the patient, it being a common trick of the malingerer or hysterical patient to heat the thermometer bulb by friction, by taking hot drinks just before the tube is applied or by utilizing a hot water bag. The highest temperature observed by the author was 110 under the tongue, and occurred in a woman of hysterical temperament suffering from spinal caries. Elevation of temperature may occur in hysteria, a fact never to be forgotten. The highest personally observed (104° F.) was obtained during an hysterical paroxysm following great emotional shock and the attack and the fever subsided almost instantly under sharp ovarian pressure. Some authorities claim that temperature may persist for weeks in hysteria and even simulate typhoid.‡ The significance of high readings is variable and certain hyperpyrexias seem to indicate little more than an intense infection associated with vigorous constitutional resisting power, such cases being seen in connection with lobar pneumonia, in which a temperature of 105° F. is neither uncommon nor necessarily of fatal import. Extremely high readings, *if long sustained*, indicate a most serious prognosis and a sudden rise sometimes precedes death. Subnormal temperatures may be observed in those suffering from collapse, chronic exhausting or wasting dis- Malingerers. Hysteria. High fever. "Agonal" temperature. Subnormal temperatures.

* Many cases of chronic larval appendicitis yield similar temperatures.

† A recent investigation of nurses' temperatures undertaken by the author in a local hospital has further strengthened this opinion.

‡ Until we know more of the real nature of hysteria major and find fewer examples of its confusion with organic ailments it might be safer to assume that *long continued* fever *excludes* hysteria.

eases, insanity (especially melancholia), chronic incompensated heart disease,* etc. Oftentimes the *surface temperature is subnormal and the rectal high, notably in cholera and tuberculosis.*

Febrile Types.—Fortunately for the clinician, the fever in many of the acute diseases pursues a more or less characteristic course or bears a peculiar relation to the appearance of other symptoms of the disease. Fevers may be placed under three general heads:—

1. Intermittent. 2. Remittent. 3. Continuous.

Intermittent.

Remittent.

Continuous.

Indeterminate.

An *intermittent fever* however far it may rise, drops to or below normal during some part of the 24 hours (see fig. 5). A *remittent fever* shows marked remissions during the 24 hours; while in *continuous* fever the remissions are slight. These are but the types and one must understand that a continuous fever may at some stage show remissions, or a remittent fever encroach upon the intermittent; indeed the temperature charts covering long periods may in such diseases as tuberculosis show every type of fever and yet be predominantly intermittent. The intermittent fever of this disease is often given the name *hectic.* Roughly speaking one may say that when the maximum daily variation in a moderate or severe fever is less than 2 degrees it is continuous, when more than 2 degrees and yet with an average minimum distinctly above the normal it is remittent; and that any fever showing a tendency to periodically fall to or below normal, is intermittent. The maximum stage of fever is known as its *fastigium,* the term being applied especially to the fevers of the continued or remittent type and to the disease as well as to the fever itself. Its opposite is called the period of *defervescence or decline.*

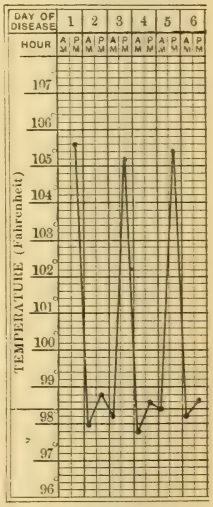


Fig. 5. Clinical Chart of Ordinary or Tertian Malaria, showing three febrile paroxysms occurring on alternate days. Typical and extreme intermittent temperature. (From Wilcox's "Fever Nursing.")

Fastigium.

Defervescence.

Diagnostic Import of Fever.—Putting aside the rare cases of hysterical temperature and excluding deception and faulty technique, one may say that fever is primarily of value in *proving the existence of some organic ailment,* a matter of no little importance in dealing with neuras-

* A temperature under 90.5 F. was recently observed by the author in an *ambulatory* mitral case.

thenics and malingerers. Furthermore, save in children, it indicates infection or auto-intoxication, acute or chronic, or irritation of some portion of the central nervous system. The absence of fever however does not rule out any one of these conditions, for there is no disease ordinarily febrile that may not though in the rarest instances exist without fever or even with sub-normal temperature. It is best exemplified in the case of afebrile typhoid, occasional pneumonias, especially those of old age, and certain cases of general peritonitis. Fever seems to guage the reciprocal action of toxæmia, infection and individual resisting power. By far the greater number of fevers are due to the action of pathogenic germs and their toxines, yet many are due to other toxic substances or even those generated by the body itself as in the

Afebrile cases.

Measures toxæmia and resistance.

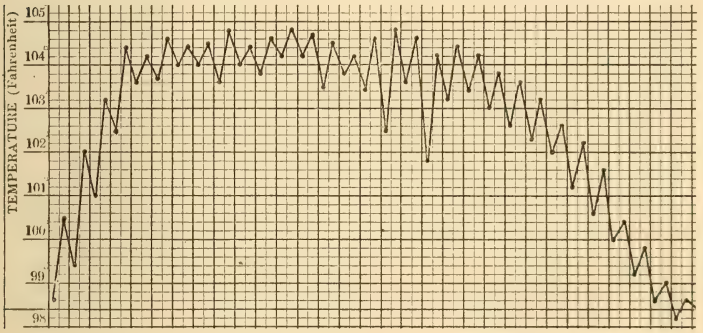


Fig. 6. A Typhoid Fever Chart showing temperature typical and unmodified by treatment. (Rare.) It serves to illustrate the continuous and remittent type and lysis and shows the so-called "step-ladder" fever of the first week. (Wilcox).

ptomaine poisoning due to the ingestion of deleterious food products and the curious auto-intoxication associated with deficient or faulty metabolism as illustrated by the terminal stages of diseases of nutrition, of malignant disease, gout, and certain of the severer types of anæmias. Fever of central origin may be encountered in cerebral embolism, thrombosis, apoplexy, brain tumor, and direct injury, or even by the changes incident to heat-exhaustion and sunstroke. Malnutrition is sometimes associated with fever, usually of a mild grade, which ordinarily occurs after severe exhausting ailments associated with much wasting of tissue and loss of strength.*

Auto-intoxication.

Cerebral.

Nutritional.

Occurring under such conditions it is *more likely* to be due to a

*The so-called "hunger" temperature may be most misleading in convalescent typhoids who have been too long starved.

smouldering tuberculosis or a complication, though it cannot be denied that cases of exhaustion temperature do occur.

Consider onset type and cause.

THE PHENOMENA OF FEVER.—One must first determine whether the fever came on suddenly or gradually, whether it was associated with chill or in the child convulsions, for how long it has endured, whether it is to be classified as distinctly intermittent, remittent or continuous, and furthermore, whether it is associated with any history of infection or with the appearance of an exanthem. If the latter be the case its exact time of appearance and its relation to any rise or fall in the temperature must be noted.

Dry and hot vs. moist.

Condition of the Skin.—In most fevers the skin is dry and palpably hot. Occasional cases depart from this rule and certain diseases such as acute rheumatism and cholera almost invariably violate it.

Digestive Organs.—Nausea, vomiting or diarrhœa (initial, persistent or terminal), constipation, loss of appetite and a coated tongue are present in varying degrees in all severe acute febrile ailments.

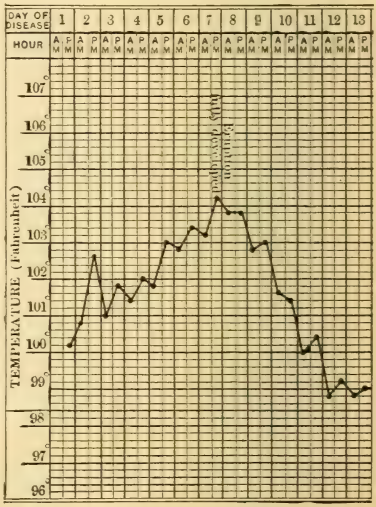

DAY OF DISEASE
1 2 3 4 5 6 7 8 9 10 11 12 13
HOUR
A M P M
TEMPERATURE (Fahrenheit)
107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°
pulse
temperature

Fig. 7. Clinical Chart of Measles showing defervescence by lysis beginning when the eruption is fully developed. (From Wilcox's "Fever Nursing.")

Aching head and legs.

The Nervous System.—Headache, and pain in the limbs are present in nearly all cases of fever, though often only at its onset. The mind may be clear or clouded and delirium may be present at the onset only, or for long periods, continuously or only at night. This may take the form of transitory illusions or hallucinations, may be violent and maniacal, mild, or of the low muttering, mumbling type of the typhoid state, perhaps with open but unseeing eyes (coma vigil). Persistent delirium of the violent or low muttering type is of unfavorable prognostic significance. Children show an especial tendency to delirium even in fever of moderate intensity, whereas, it is rare in adults save in the most severe types of infection, in the intoxications or in central disturbances.

Mental condition.

In children.

Acute Febrile Delirium.—(Bell's mania). This rare condition peculiar to women and of unknown causation, is initiated by shock or other emotional strain, and may be associated with the convalescent state. *It consists essentially of a violent, continuous, exhausting and maniacal delirium, associated with high fever and a typhoid state.* No specific pathologic findings are present after death and the disease may readily be confounded with the acute maniacal delirium occasionally present at the onset of pneumonia, typhoid and uræmia. It is rapidly fatal, running its course in from one to three weeks. The differential diagnosis is one of exclusion.

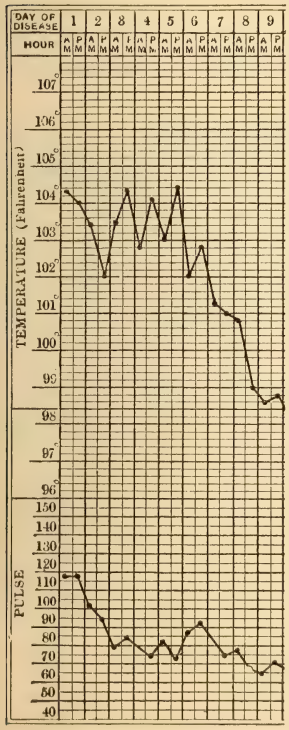

DAY OF DISEASE
1 2 3 4 5 6 7 8 9
HOUR
A M P M A M P M A M P M A M P M A M P M A M P M A M P M A M P M
TEMPERATURE (Fahrenheit)
107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°
PULSE
150 140 130 120 110 100 90 80 70 60 50 40

FIG. 8.—Clinical Chart of a Yellow Fever Patient showing the pulse typically slow in comparison to the height of the temperature. (From Wilcox's "Fever Nursing.")

The Pulse and Respiration.—Both pulse and respiration bear a definite relation to the temperature curve. The increase in pulse rate is ordinarily from 8–10 beats for each degree of fever and a departure from this rule is especially noted in typhoid fever, yellow fever and lobar pneumonia, and favorably affects prognosis in these diseases. The heart muscle suffers to a variable degree in all infections and intoxications and must be carefully watched for signs of weakness, degeneration or valvular inflammation, this being especially true as regards the valves, heart muscle and pericardium in attacks of acute or subacute rheumatism and in diphtheria. The pulse of high fever of the so-called sthenic type is not only rapid but full and bounding. In asthenic fevers it becomes soft, weak and in the typhoid state, dicrotic. In acute and chronic affections of the kidney it is of high tension and such a pulse should always direct attention to the kidneys, and calls for examination of the urine. In inflammation of the peritoneum it is small, hard, and wiry in feel.

Normal ratio.

Typhoid and pneumonia.

Heart muscle.

Sthenic vs. Asthenic fevers.

Renal pulse.

Exanthems.—The accompanying charts show the peculiarities of fever in certain diseases. In *measles* the temperature recedes 24 hours

or thereabouts after the onset, rising to reach its highest point with the coming of the rash on the fourth or fifth day.

German Measles.—(Rötheln). In rötheln the eruption appears on the first day or more rarely the second, and is usually the first thing noted. The fever rising much the same but usually less than in measles.

Scarlet Fever.—In scarlet fever the rapidly spreading rash appears on the first or beginning of the second day following an abrupt onset with high fever.

Varicella.—(Chickenpox). In varicella the rash appears within 24 or 36 hours associated with trifling fever.

Variola —(Smallpox). In variola the violent onset, usually with chill and high fever is followed by a marked remission during which the rash appears in its papillary stage. During its subsequent changes the temperature rises, reaches its maximum with the purulent transformation of the vesicles and then gradually subsides. The diagnostic value of these differences in the relation of body temperature to the eruption is readily appreciated.

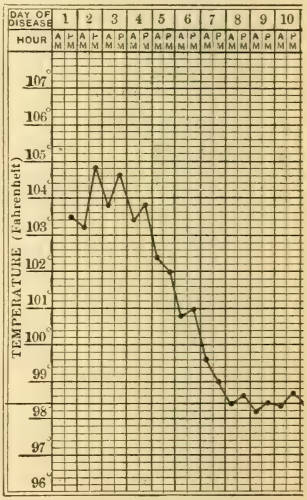


Fig. 9.—Clinical Chart of Scarlet Fever. (From Wilcox's "Fever Nursing.")

The Mode of Onset and Termination.—The more severe of the acute infections are usually preceded by a decided "chilliness" or an actual chill in adults* and in children by either chilling or convulsions. In several diseases however the onset is insidious and without marked initial phenomena and in senile cases this may be true of ailments ordinarily frank. The termination of fever is in most cases a gradual recession (lysis) but certain ailments of which lobar pneumonia is the type terminate suddenly (crisis). The *critical* phenomena vary somewhat but usually the temperature falls to or below normal within 24 hours, often yet more abruptly, suffering is replaced by relative comfort, profuse sweating and sometimes diarrhœa occur and the contrast is often most dramatic. A sim-

Frank onset.

Insidious onset.

Senile cases.

Lysis and crisis.

* A severe chill is certainly less generally observed even in pneumonia than the student is usually led to believe.

ilar fall in temperature in diseases not associated with true crisis points usually to a serious complication, in typhoid, for example, to a hemorrhage or perforation. In other cases it may be the antemortem pseudo-crisis. In pneumonia, a pseudo-crisis of marked degree is often followed by renewal of fever, the true crisis occurring ordinarily on the day following. In one case of migratory pneumonia observed by the author, lobe after lobe of the lungs went through a typical pneumonic cycle, the critical fall being promptly followed by chill, renewed fever and the usual signs of consolidation in the adjacent lobe.*

Pseudo-crisis, ante-mortem recession.

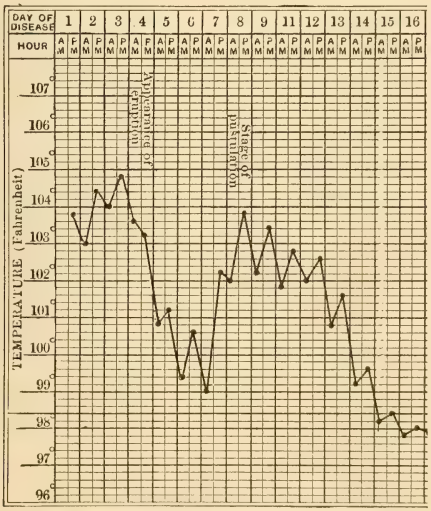

DAY OF DISEASE
1 2 3 4 5 6 7 8 9 11 12 13 14 15 16
HOUR
A M P M
TEMPERATURE (Fahrenheit)
107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97° 96°
Appearance of eruption
Stage of pustulation

Fig. 10.—Clinical Chart of Smallpox showing fall of temperature upon the appearance of the eruption and its rise upon the incidence of the stage of pustulation. (From Wilcox's "Fever Nursing.")

COMA AND ITS CONGENERS.—Coma covers any state of prolonged unconsciousness from which a patient can be aroused only partially, temporarily, with difficulty or not at all.† The dividing line between different states of unconsciousness and profound sleep is ill defined and one merges into the other. The term *coma vigil* is applied to the peculiar complete or semi-unconsciousness of certain cases of typhoid fever associated with an open eyed, low muttering delirium.

Coma vigil

*Case of Dr. H. P. Ritchie.

†In most instances true coma is associated with a relaxed jaw, stertorous breathing and dry tongue, e. g. morphine poisoning or apoplexy.

Stupor and Lethargy.—Both represent sleep or sleepiness, the former profound as in alcoholism, the latter a mere drowsiness as in the premonitory stage of freezing. Assuming that a patient is seen for the first time on the street, in the ambulance, hospital or the home, and is comatose or stuporous, the difficulties in diagnosis are so great as often to baffle the diagnostician. Yet many avoidable errors occur through lack of proper method and ordinary knowledge and carefulness. *The conditions under which an attack occurs* should be first sought through some friend, relation or bystander. *The appearance* of the patient at the time of the attack, his movements and the direction and force of his fall are also to be considered, oftentimes his habits or even his previous health may be ascertained. *The state of the atmosphere* may at once suggest heat-exhaustion or sunstroke. Inquiry must be made as to *what the patient has taken into the stomach*, having in mind the possibility of ptomaine or drug poisoning, or of sudden deaths or severe prostration following the overloading of the stomach in victims of the diseases of the heart and arteries. In public services the history is often entirely lacking or untrustworthy and the physician is thrown upon his own resources. He has to consider *epilepsy*, *malingering*, *strychnia*, *uræmia*, *apoplexy*, *diabetic coma*, *poisoning by opium*, *chloral or other narcotics*, if delirium is present, *belladonna*, if convulsions, *strychnia*, and investigates:—

Cause often obscure.

Attendant conditions.

Essential data.

Temperature and humidity.

Ingesta.

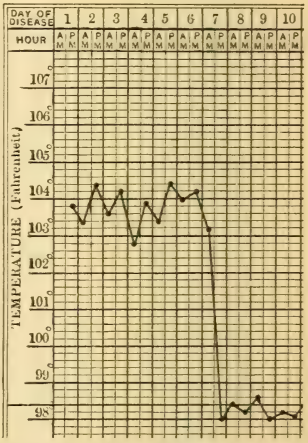


Fig. 11.—Clinical Chart of Acute Lobar Pneumonia showing crisis upon the seventh day of the disease. (From Wilcox's "Fever Nursing.")

Data.

(a). **The Eyes.**—Are the pupils equal or unequal, contracted or dilated? Is the conjunctival reflex present, do they react to light, and is there resistance to the separation of the lids? *Unequally dilated pupils* (the larger usually on the side of the lesion) suggest cerebral hemorrhage or general paralysis of the insane. *Contracted pupils* suggest opium poisoning, or pontine hemorrhage; in *uræmia they are usually dilated* and in all these conditions they fail to respond to light and lack the conjunctival reflex. The same is true of cocaine and the later stages of chloral poisoning. *If convulsions are present* with fixed,

Pupils.

dilated pupils and absent conjunctival reflex, epilepsy or poisoning by strychnia, or some other convulsant is at once suggested. In *alcholism* the pupils may be dilated, but usually respond to light. *Hysteria and malingering are as a rule readily detected by the prompt response to light, and the resistance almost invariably offered to the separation of the lids by the physician.* An examination of the background may reveal an *albuminuric retinitis* in uræmia or *choked disk* in brain tumor.

Epilepsy strychnia.

Alcoholism.

Hysteria and malingering.

(b). **Color.**—In alcoholism the face is usually flushed, in cerebral lesions it may be flushed but is usually cyanotic, in uræmia there are ordinarily pallor and cyanosis, and this latter may be excessively marked in acetanilide poisoning.*

(c). **The Degree of Unconsciousness.** The conjunctival reflex should be tested in all cases. Sharp pressure over the ovarian region may resolve doubt in hysteric females, being often accompanied by a complete or partial return to consciousness. The malingerer and even the alcoholic individual can seldom resist moving, if such a stimulus as the sharp pinching of the skin of the inner surface of the arm is applied, or the police method of pounding the soles of the feet followed. In the other and more serious forms of coma, insensibility is as a rule complete.

Ovarian pressure in hysteria.

Alcoholism.

(d). **The Nature of Convulsive Movement if Present.**—In cerebral hemorrhage conjugate deviation of the eyes and head is most common. In the comatose terminal stage of strychnia poisoning both head and trunk are involved, opisthotonos or emprosthotonos are likely to be present and between the attacks the patient may be conscious and rational.† In *tetanus* the convulsive seizures are likely to be limited to the neck and jaw, and complete relaxation between seizures does not usually occur. In *epilepsy* the sequence of the seizures, i. e. clonic followed by tonic convulsions and the absence of opisthotonos are suggestive. The malingerer is foolish to attempt convulsions on account of the difficulty of imitation. In *hysteria*, though often strikingly epileptiform or tetanic they are usually irregular and associated with the typical hysteric facies, and though the lips may be bitten, the tongue is not, whereas in true epilepsy biting of the tongue and incontinence

Apoplexy.

Strychnia poisoning.

Tetanus.

Status epilepticus.

Malingerers.

Hysteria.

* In a puzzling case seen by Dr. Haldor Sneve profound coma and extreme cyanosis were explained by the fact that the patient had used as much as one or two pounds of a preparation of bromo-seltzer = 40–80 grs. of acetanilid daily for several months. The case recovered.

† Neither in strychnia poisoning nor tetanus does complete, persistent unconsciousness supervene until the terminal stage and often not at all.

of urine during the seizures are important signs. In hysterical tetanus the eyes are usually closed during a seizure and emotional utterances, sobbing, and crying are prominent. The peculiar premonitory exhilaration and hyperacuteness of the special senses observable in strychnia poisoning are absent in hysteria and the onset of spasm is atypical.

(e). **Paralysis.**—The limbs should always be handled to ascertain if paralysis be present in any member, the peculiar lack of all muscle tone being usually readily determined and paralysis of the muscles of the eye and face easily noted. If paralysis of the extremities be associated with marked atrophy it will of course suggest an old lesion which may or may not be related to the patient's present state. Froth about the lips, if blood stained, points to epilepsy, but is common to many of the convulsive states attended by unconsciousness. Malingerers are usually obliged to use soap for this purpose.

Muscle tonus.

Atrophy.

Frothy lips.

(f). **The Pulse.**—In cerebral compression the pulse is slow, in uræmia its high tension may be suggestive. *In malingerers and hysterical persons it is the pulse of exertion*, rapid, full and bounding and in other conditions it is, as a rule, of little value.

High tension.

(g). **The Temperature.**—In all cases the rectal temperature should be taken and particularly in the cases apparently alcoholic in their nature. One of the commonest mistakes on the part of ambulance and police surgeons results from the failure to remember that pneumonia and cerebral lesions are extremely common in the drunkard, and may occur during the unconscious stage. As before stated the presence of fever does not absolutely exclude hysteria and it is present in cerebral hemorrhage, the surface temperature being usually a degree or more higher on the paralyzed than on the non-paralyzed side.

Fatal errors.

(h). **The Breath.**—An alcoholic odor though suggestive does not prove drunkenness. The so-called uræmic breath is reasonably characteristic, but closely approximated by that encountered in other conditions, and is a disagreeably aromatic odor that must be learned by actual experience. The sweet breath of the diabetic is far more characteristic and often extremely penetrating.* Certain poisons like laudanum, carbolic acid, hydrocyanic acid, ether and chloroform yield a characteristic odor.

Uræmic odor.

Diabetic breath.

Poisons.

(i). **The Examination of the Urine.**—At the earliest moment a

* The author not long since detected it in passing upon the open street a little babe supposed to be in perfect health but suffering from terminal diabetes, as determined by the family physician upon notification.

specimen of urine should be removed by catheterization and carefully examined with reference to contained solids, albumen or sugar.

Conclusion.—In conclusion it must emphatically be said that but few of the differential points given are absolutely distinctive, and there is no condition that more greatly taxes the skill and common sense of the practitioner. If the breath of a patient smells of laudanum, the pupils are contracted, the breathing stertorous, the skin cyanotic, and clammy, and particularly if a receptacle that has contained laudanum is found empty, the proof is sufficiently conclusive. Stertorous breathing, flushed countenance, slow hard pulse and the evidences of paralysis are sufficiently characteristic of a cerebral lesion. On the other hand, *one often meets with conditions in which he is misled by a reversal of ordinary symptoms, contradictory or confused findings and a failure on the part of the ailment to observe the man-made laws of differential diagnosis.*

Diagnosis often impossible.

PAIN.—*Pain as a symptom is purely subjective and may be true or false, localized or diffuse, momentary, temporary or persistent and vary greatly in intensity.* Its differences are indicated by such descriptive terms as dull, aching, sharp, acute, stabbing, boring, gnawing, lancinating, shooting, colicky, radiating, diffuse, etc. It may be deeply seated or superficial, and may or may not be associated with tenderness due either to actual inflammation or to hyperæsthetic areas. Occasionally the site of pain is far distant from its source, and indeed certain referred pains may be dangerously misleading, for example, the author has observed a case* in which an aneurism of the *left* iliac artery caused pain over the appendix, others in which appendiceal pain was referred to the left side, yet others in which the pain of gall stone colic appeared in the region of the heart, while nothing is commoner than for the pain of pleurisy to be referred to the surgical regions of the abdomen along the intercostal nerve terminals.

Subjective and variable.

Referred pain.

The Character and Seat of Pain in Relation to Diagnosis.—The pain of an acute inflammatory disease is usually associated with fever, and often with localized tenderness, the degree of either depending upon the nature of the affected structure, the individual susceptibility and resistance, and the extent and situation of the inflammatory process. In surgical diseases of the extremities, for example, the pain is ordinarily associated with distinctly localized tenderness and is of great value as a symptom. On the other hand a pneumonia may exist with little or no pain or by involvement of the pleura produce excessive pain. *In*

Factors in severity.

Variable.

* Through the courtesy of Dr. Archibald McLaren.

Painless inflammation.

Movement and pain.

Relief by exudate.

Radiating.

Dull.

Colicky.

Gnawing and boring.

any disease of a viscus inflammation of the substance is almost painless until extension to the internal or external layers occurs. Pain is likely to be distinctly related either to *bodily movement* in general, or to *functional activity* of the part affected, for example, friction of inflamed surfaces causes respiratory pain in pleurisy, while in acute rheumatism the slightest movement of the affected joint is distressing. In all such instances nature seeks to relieve the condition by pouring out a fibrinous or serous exudate which fact explains the partial or complete relief of pain in fully developed cases of pleurisy and pericarditis. *Radiating pain* is most characteristic of neuralgias and neuritis, and is recognized by its tendency to follow the known distribution of nerve trunks and by association with marked tenderness over certain areas. *Dull pain* may take the form of a general aching as in the acute infectious diseases, or may attend many of the chronic low grade inflammations, particularly such as affect the mucous membranes and the parenchymatous structures. Acute inflammation or irritation of certain mucous membranes may be attended with severe pain, as for example, that of renal colic. *Gnawing or boring pain* is frequently encountered in caries of the spine, aneurism of the aorta, carcinoma of the stomach and inflammation of bone. It is also met with in some cases of old gastric ulcer, in gout, gall stones and renal calculus.

Paroxysmal Pain.—Gall stones, renal colic, colon colic, locomotor ataxia, neuralgia and angina pectoris furnish the best examples of paroxysmal pain; nearly all radiating pains are distinctly paroxysmal.

Colic.—The term colic applies chiefly to paroxysmal abdominal pain and includes that of biliary or renal calculus, appendicitis, lead poisoning, floating kidney (Dietl's crises), mucous colitis, spastic constipation, strangulated hernia and abdominal aneurism. Many of these attacks were formerly covered by the term "gastralgia," but the modern surgeon has proven that an actual gastralgia (neuralgia of the stomach) is so rare as to be almost negligible. Cases of simple transitory colic due to acute indigestion, are common in both children and adults, but the utmost caution must be observed in the interpretation of an *apparently* simple abdominal pain, particularly if unduly persistent and associated with tenderness on deep pressure.

Gastralgia a misnomer.

Onset.

McBurney's point.

Appendicitis.—Many cases begin with *general abdominal pain* of a *colicky* type and 12 or 24 hours may elapse before it becomes distinctly localized at a point of *maximum tenderness* in the area lying midway between the anterior superior spine and the umbilicus (McBurney's

point). The tenderness may be apparent before the pain becomes localized and both are simulated by certain cases of renal colic in which a small calculus has become engaged in the lower portion of the ureter. Nausea and vomiting at the onset, with fever assist the diagnosis. *Chronic appendicitis* often remains problematic or is only revealed by operation, but usually it may be determined by the *history*, by temporary *exacerbations*, or by *actual palpation* of the affected member. The disease is frequently associated with symptoms of chronic indigestion of an indeterminate type. See page 288.

Ureteral colic.

Chronic cases.

Renal Colic.—This *sudden, intense* and *agonizing paroxysmal pain* may result from stone or gravel, either in the kidney pelvis or passing downward through the ureter. There may be *grunting* and *straining expulsive movements*. It has a *maximal point over the kidney* posteriorly *and tends to shoot downward* over the course of the ureter into the groin along the inner portion of the thigh and into the testicle, which is oftentimes retracted during the paroxysms. Unlike appendicitis, *it is usually afebrile*, and *dysuria* and *frequent micturition are associated with albumen, blood and gravel or actual calculi in the urine. Nausea* and *vomiting* are common and in two instances the author has known of narrow escapes from operation for appendicitis apparently because of temporary block by calculi at the lower end of the ureter. In renal colic *tenderness* is usually found over the kidney posteriorly, often anteriorly, and is best elicited *by deep pressure over the 12th rib, or according to the author's experience just beneath it, at the end of deep inspiration.*

May simulate appendicitis.

Tenderness.

Dietl's Crises.—May be associated with pain of the same type, with more or less tenderness over the region of the movable or floating kidney, but more closely simulates gall stone colic than renal colic so far as pain distribution is concerned. *The incidence and severity of attacks bear but slight relation to the degree of kidney mobility.*

Simulates gall stone colic.

Gall Stone Colic.—Is indicated by *paroxysmal pain in the epigastrium or right hypochondrium, radiating both upward and downward, but chiefly upward*, being often felt in the right shoulder joint and under the right scapula, and is associated with *tenderness on deep pressure* over the gall bladder at the end of deep inspiration. It is accompanied by nausea or vomiting and may or may not be associated with jaundice. See page 305.

Colon Colic.—In association with mucous colitis and with chronic spastic constipation, a paroxysmal attack may occur quite as severe as that of many renal colics. It is left sided, the maximum point is over the region of the kidney, posteriorly, but ordinarily it lacks the typical

Often severe.

thigh and groin radiation and is either wholly or in a considerable measure relieved by emptying the bowel with hot enemata.

Relieved by pressure.

Lead Colic.—*A paroxysmal, diffuse, persistent and recurrent* pain associated usually with obstinate constipation and relieved rather than increased by pressure, should lead one to search for a "blue line"* on the gums, or for some source of lead poisoning.

Lead line.

The curious paroxysmal pain associated with the crises of locomotor ataxia and the obscure and rarely diagnosticated pain of arterio-sclerotic abdominal crisis or abdominal aneurism must always be borne in mind.

Tabes, aneurism.

Colic due to Gastric Ulcer.—*Violent paroxysmal pain* may be encountered in connection with gastric ulcer and the pain is usually *epigastric, deep seated* and often seems to pass through to the back. During acute attacks it *may* be relieved by pressure, more often however, as in the chronic form, any pressure is distressing. It is usually associated with dyspepsia and anæmia and often a history of hæmatemesis assists the diagnosis. In nearly all forms of abdominal colic the onset is sudden, with few or no prodromata and nausea and vomiting almost invariably occur. See p. 262.

Pressure tenderness.

General rule.

Acute Intestinal Obstruction.—Acute obstruction is characterized by paroxysmal pain, becoming continuous, and ultimately by fecal vomiting. *The obstinate constipation is often preceded by misleading evacuations from that portion of the bowel lying below the point of obstruction.* If the obstruction involve the colon there is less vomiting, marked abdominal distension and relatively slight indicanuria. In lower ileum or cæcal obstruction, the distended coils may form a ladder pattern; if in the sigmoid or rectum the descending, transverse and ascending colon may be projected by distension as a visible horseshoe curve. *An actual tumor* is rare save in *fecal accumulation* or *intussusception*. The latter occurs in three-fourths of the cases at the ileo-cæcal junction and most often in infants, rarely in young adults, with bloody mucoid stools and tenesmus.

Constipation.

Symptoms vary with site.

Tumor.

Stools.

Volvulus.—Obstruction due to axial twist seldom occurs in youth, most often involves the sigmoid flexure or the region of the cæcum and is associated with marked meteorism, rigidity and tenderness. Antecedent constipation and flatulence usually exist and in sigmoidal cases the obstruction may be so low as to make the condition evident when an attempt is made to introduce a large enema.

Adults chiefly.

Sigmoidal cases.

Strangulation.—In the adult this is a most frequent source of acute obstruction and is most frequently caused by old adhesions, various

*See also "gums," p. 23.

openings and pockets, slits in the mesentery and omentum, rupture of the diaphragm, various hernias, and adhesions of the tip or persistence of, Meckel's diverticulum.

Intestinal Paralysis.—In connection with abdominal operation and as a result of sepsis and peritonitis, there may be an entire absence of intestinal activity leading to what is essentially an acute obstruction associated with general distension and recognized by the absence of normal sounds upon auscultation.

Post operative.

Auscultatory sign.

Fecal Impaction.—A fecal mass gradually accumulating in an atonic bowel may be found either at the cæcum or sigmoid flexure and may wholly occlude the lumen or contain a central channel through which a certain amount of material may pass. A localized peritonitis with pain and tenderness may or may not be present and the mass may be *felt* in the absence of extreme distension. The condition most frequently occurs as a result of neglect of the bowels, or in nervous conditions, in the aged, apathetic, hysterical or insane.

Site.

Complete or partial.

Tumor.

Of tumors, strictures and occlusion by foreign bodies little need be said. Cicatricial contraction results usually from old ulcerations, whether syphilitic, tuberculous, dysenteric or simple; less often from adhesion. Occlusion by tumor may be due to development of local growths, direct pressure, or associated adhesions.

Ancient ulcerations.

General Comment.—Early and correct diagnosis is of importance in connection with intestinal obstruction, hence the leading features should be kept closely in mind. *The higher the obstruction the less is the meteorism and associated tumors point to an actual growth, intussusception or fecal impaction.**

Meteorism and tumor.

Intussusception *is most frequent in infancy and childhood, and the tumor is usually in the ileo-cæcal or appendiceal region or in the sigmoid flexure.* In the latter, and in volvulus the lack of fluid capacity of the lower bowel may be an important aid. *Volvulus is most common in the fourth decade. Marked tenesmus and bloody stools are most frequent in intussusception,* the passage of *ribbon like* or greatly *narrowed cylindrical stools* suggests *obstruction,* but may occur in cases of fecal impaction possessing central calibration or even be due to hemorrhoids or rectal spasm. Further, though acute constipation *with retention of both gas and fecal matter is the cardinal sign of complete obstruction, the unoccluded lower portion of the bowel may be able to eject its contents.* Foreign bodies of any kind may cause obstruction and are seldom suspected unless a clear history is obtained. Most frequently the

Test by enemata.

Stools.

* Such impaction may be enormous.

victims are children or mentally defective patients. The persistent administration of large doses of bismuth and magnesia may cause obstructive masses, and amongst other rare conditions are bunches of intestinal worms, masses of hair, and gallstones which have made their way by ulceration from the gall bladder to the bowel.* *Fecal vomiting may require 24 or 48 hours to develop its characteristic brownish color and specific odor. Obstruction may be so low down as to be felt by rectal palpation and in descending colon and sigmoid obstruction* the *sphincter* may be relaxed. Fever is present, but usually slight, thirst is excessive and in a few days collapse appears and terminates promptly in death.

Drug obstruction.

Worms.

Fecal vomiting.

Rectal palpation.

Anæsthesia.

Diagnosis by Inflation and Liquid Injections.—An anæsthetic is necessary and should be pushed to full relaxation. Rectal palpation should be followed by the introduction of a long tube (Kelly's tube) or oftentimes the whole hand may be inserted.† For the injection of water which is in every way superior to air, the patient should be placed in the knee-chest position, the warm water injected under gentle pressure and its passage determined by both percussion and auscultation. A normal colon should hold about 8 litres (quarts) and the ileocæcal valve *usually* blocks any flow into the lesser bowel. In children the injection may be varied according to age, though even an infant can take from 1 to 1½ litres.

Normal colon capacity.

Comment.—*The following points should be remembered in connection with abdominal pain:*—Jaundice when present suggests gall stone colic, but its absence is of little consequence as disproving that diagnosis. Fever is entirely absent as a rule in colon colic, lead colic, gastric ulcer and abdominal aneurism. It is almost invariable in acute appendicitis, frequent in gall stone colic, more often absent in renal colic. The examination of the urine should never be omitted and may reveal the blood and albumin of kidney involvement, the high grade indicanuria of ileus, the pale color and increased quantity suggestive of hysteria or the actual stone particles of renal calculus. The examination of the blood may show leucocytosis due to appendicitis, or the basophilic granulation of red cells suggestive of lead poisoning.

Jaundice.

Fever.

Urine.

Necessary data.

HEADACHE.—The following points should be considered, viz.:—(a) *location*, (b) *character*, (c) *severity*, (d) *distribution*, (e) *time of occurrence*, (f) *duration*, (g) *local tenderness*, (h) *hereditary predisposition*, (i) *the effect of medication* (in syphilis, anæmia, malaria, etc.) and the *underlying cause*.

* The only way that a large gallstone can escape.

† This procedure is not free from danger.

Anæmic headache *affects either the forehead, orbits, vertex, or occiput.* It is usually moderate and associated with a sense of pressure, but in *extreme* chlorosis is occasionally so severe as to resemble the headache of meningitis. Ordinarily mild.

Bilious or Dyspeptic Headache.—This toxæmic variety includes as etiologic factors or associated conditions, lithæmia, jaundice, the dyspepsias and constipation. It is irregular in duration and occurrence, frontal and congestive in type and associated with nausea. There is a subjective sense of pulsation; a tendency to nausea; it is increased by motion and by stooping, and more or less promptly relieved by emesis and catharsis. Toxæmic. Frontal and congestive.

Brain Tumor.—This causes persistent headache, usually increased at night, associated with nausea and vomiting, and sooner or later with optic neuritis. See p. 578. Optic neuritis.

Brain Abscess.—This may cause severe and persistent headache resembling that of brain tumor but is usually accompanied by a septic temperature curve and optic neuritis is usually absent. See p. 586. Papillitis rare.

Eye Strain Headaches.—May be either occipital, orbital or frontal, are usually associated with use of the eyes for close work, or in certain cases for distances, relieved by sleep and frequently associated with conjunctivitis, tenderness of the eyeball or eye muscles and sometimes with blurred vision.

Drug Headaches.—The overuse of drugs may lead to headaches which have no distinguishing features. A dull throbbing headache associated with ringing in the ear often follows the administration of a salicylate or quinine in full doses.

Bright's Disease.—(See uræmic headaches) p. 350.

Arterio-sclerosis.—The cause is suggested by rigid temporals or radials and by the fact that the headaches are often relieved by the administration of such a drug as nitroglycerine. They are seldom severe though often persistent; are frequently associated with impaired memory and intellection and transient vertigo and *are often forerunners of cerebral hemorrhage.* Suggestive facts.

Malaria.—Both dull, persistent headache and obstinate neuralgia may be encountered in connection with malaria. Unless it is distinctly periodic, the only characteristic feature is its subsidence under appropriate drug treatment (quinine). Therapeutic test.

Migraine.—(Sick headache, hemicrania). Heredity plays a large part in migraine which is regarded by many as an epileptic equivalent. Females are most frequently attacked and a considerable proportion of

Various causes.

the cases (30%) commence in childhood (5th to 10th year). Aside from family taint, conditions such as eye strain, adenoids, carious teeth, pelvic disease, gastric disorders, and overexcitement, overstudy and dietetic errors are most important. The author has seen a number of cases of the most extreme and persistent type associated with a renal insufficiency without other signs of the chronic nephritis with which headaches of this type are so frequently associated. Periodicity may be marked even to the hour of the day, or it may bear a definite and invariable time relation to menstruation.

Nitrogen retention cases.

Symptoms.—The attack appears usually in the early morning, often preceded by localized numbness and tingling, hippus, vertigo, visual paræsthesias (flashes of light, fortification figures), cramps, spasms, or even aphasia associated with mental exhaltation or depression. *The headache is unilateral, usually right sided and sharp, stabbing or boring in character.* Nausea and vomiting are common symptoms, movement, light and sound increase it, it may become bilateral and extensive and pallor or flushing may be marked. In a periodic case recently observed in a young child, the hour of waking, transient aphasia, extreme restlessness, nausea and vomiting ushered in a typical hemicrania, which lasted for but two or three hours. Usually the duration is one, sometimes two or three days, a night's sleep ordinarily bringing relief. In children the *possibility* of a developing meningitis or brain tumor must be remembered.

Hysteria.—*There is no headache characteristic of hysteria* and "clavus" (pain at the vertex as if a nail were being driven into the skull) has never been encountered by the author.

Neurasthenic Headache.—Usually mild and indeterminate, frequently occipital, the important characteristic of this form is its morning maximum and the tendency to disappear under physical exertion and mental preoccupation.

Nasal Headaches.—This form is characterized by pain originating between the eyes and sometimes running backward to the occiput,

Cause usually clear.

greatly increased by coughing, sneezing or stooping, worse in the morning and evening, and relieved by any measures reducing the congestion of, or relieving the pressure upon the nasal structures affected. These are essentially turbinal or frontal sinus headaches resulting from congestion or hypertrophy of the turbinate bones or diseases of the sinus.

NEURALGIA.—The pain of severe neuralgia is acute, radiating, distinctly localized and associated with superficial points of tenderness. Its

characteristic feature lies in its definite relation to known nerve trunks, their points of exit and their distribution. It may affect any portion of the body, though most common in the head, and is usually the expression of toxæmia, though frequently initiated by extreme fatigue, particularly when associated with exposure to cold and damp. Severe attacks are remarkable for persistence and intractibility under treatment, a tendency to recurrence, often periodic, and to chronicity. The tender points in neuralgia represent points of nerve trunk emergence, their entrance into muscles, or their terminal filaments. Maximum tenderness is usually easily determined and widely diffused sensitiveness of the most extreme type is sometimes encountered.*

Suggestive features.

Etiology.

Tenderness.

Trifacial Neuralgia.—(Tic douloureux). *Either the ophthalmic, the upper maxillary or the lower branches may be affected, the last being the most common, the first the most severe form.* In all varieties the pain is intense, the point of tenderness marked. If the ophthalmic be chiefly affected the pain is especially severe about the region of the eye, which is often injected and tender. Lachrymation may be present, and there is marked tenderness in the supraorbital region, over the bridge of the nose, and occasionally at the occiput and upper cervical spines. *Transient blindness may occur. If the upper maxillary* is chiefly affected the maximum pain follows the upper teeth and the maximum tenderness is at the infraorbital canal. *With involvement of the lower branches* the pain radiates to the ear, and along the lower teeth, the maximum painful points corresponding to the auriculo-temporal nerve. All branches are usually more or less affected, and a severe herpetic eruption may occur along the track of the superficial nerves.

Ophthalmic.

Superior maxillary.

Inferior maxillary.

Cervico-brachial Neuralgia.—This variety frequently associated with rheumatic affections involves especially the sensory nerves of the brachial plexus. It is relatively infrequent, occurs chiefly in women, may be due to remote primary causes such as locomotor ataxia, uterine disease or carious teeth, and is characterized by the distribution of the pain and points of maximum tenderness which may be over the ulnar nerve, at the elbow, near the wrists, in the axilla, at the inferior angle of the scapula, over the deltoid muscle, or even on either side of the lower cervical spine. Herpes, anæsthesia and vasomotor disturbances may occur.

Cervical or brachial.

Cervico-occipital Neuralgia.—This affects the sensory branches of the upper cervical nerves, is often caused by cervical caries and usually

*The possibility of a renal inadequacy as indicated by insufficient excretion of urinary solids should always be borne in mind.

associated with a point of maximum tenderness midway between the mastoid process and the atlas.

Etiology.

Other Varieties of Neuralgia.—*Intercostal Neuralgia.* This common ailment occurs most frequently in those debilitated by overwork, malnutrition, toxæmia or co-existing diseases, and occurs chiefly in young adults of the female sex. It is characterized by sudden pain in the chest, of the neuralgic type, but slightly affected by respiration, yet following the intercostal distribution. The maximum point of tenderness corresponds to the exits of both dorsal and anterior branches and it chiefly affects the left side. It is ordinarily of brief duration, but may last several weeks and is of special importance in connection with angina pectoris, pleurisy and the acute pain of intra-abdominal diseases, for which it is often mistaken, the pain being sometimes referred to superficial nerve endings upon the anterior abdominal wall.

Pain and tenderness.

Duration.

A neuritis.

Herpes Zoster.—Closely resembles intercostal neuralgia, but is probably a neuritis. It is usually unilateral, rarely bilateral and is associated with an herpetic eruption following the course of the affected intercostal nerve. Its pain is atrocious *and may precede the eruption by many hours.*

Causes.

Onset.

Paroxysms.

Pressure points.

Sciatica.—(Sciatic neuritis, sciatic neuralgia). In the presence of the ordinary etiological factors, sciatica is usually initiated by exposure to cold, wet and excessive fatigue, direct injury, pressure from pelvic tumor, chronic constipation or preexisting disease of the vertebra or spinal cord. It is characterized by a sudden onset, the intense pain following the course of the sciatic nerve, and often extending well up into the lumbar region. Intervals of comparative immunity alternate with paroxysms of a most excruciating type, the pain sometimes shooting from above downwards and giving the sensation of an actual shock as it reaches the heel. Subjective numbness, tingling, and disturbed temperature sense may be present. Pressure applied at the middle of the thigh, posteriorly, sciatic notch, posterior aspect of the knee and calf, the external malleolus or the dorsum of the foot reveals the points of tenderness.

Morton's toe.

Miscellaneous Neuralgic Pains.—Pain of a neuralgic type may of course occur in any region of the body, and be confined to a single trunk or even a single terminal, thus it may be limited to the breast (*mastodynia*), finger (*digital neuralgia*), the plantar nerves (*plantar neuralgia*), or be associated with the stretching of the plantar ligaments in incipient flat foot, in which case the pain is in some instances limited to the third and fourth metatarso-phalangeal joints (Morton's foot).

Lumbo-abdominal neuralgia is characterized by pain in the back, buttocks and loins, radiating to the genital and hypogastric regions, and areas of *superficial tenderness*. It is commonly associated with general debility, chronic constipation, disease of the pelvic organs and exposure to wet and cold under conditions of fatigue. *In femoral neuralgia* the pain is limited to the front of the knee and the outer front of the thigh. In diseases of the hip joint the reflex knee pain (inner aspect) is extremely common.

Femoral type.

NEURITIS.—The distribution of neuritic pains and their general characteristics are much the same as neuralgia, though much more severe and persistent. The disease may be acute or chronic, circumscribed and limited or widespread and multiple. Distinctly inflammatory in type it tends to produce primarily and essentially a degeneration of the affected peripheral nerves. Aside from the more persistent and constant character of the pain, the exquisite tenderness on pressure is characteristic. Furthermore, there is a special tendency to marked cutaneous hyperæsthesia followed by anæsthesia and such marked changes as redness or pallor, edema or joint swelling and herpetic eruptions. In the extremities the skin often becomes characteristically glossy, and disturbances of nutrition are indicated by muscle atrophy, the reaction of degeneration, loss of dependant reflexes, changes in the nails, loss of hair, desquamation of the epidermis, etc. True swelling of the nerve trunk and erythema indicating its course may be present and if the motor fibres are affected, changes appear, varying from a sense of fatigue and slight loss of power to actual paralysis.

Severe and persistent pain.

Tenderness extreme.

Cutaneous symptom.

Trophic changes.

Paresis or paralysis.

MUSCULAR RHEUMATISM.—This common and painful ailment most often affects the lumbar muscles (lumbago), muscles of the chest and the cervical muscles (rheumatic torticollis). In all, the chief characteristic is pain on movement, which produces more or less characteristic attitudes, almost complete loss of function and voluntary rigidity. One of the most important points of distinction as between this painful affection of the muscles and neuralgia, neuritis, pleurisy, and such other affections as it may simulate, lies in the fact that tenderness of the muscles themselves when grasped is a prominent feature, furthermore, the pain is distinctly related to muscular movement, and is absent or greatly ameliorated when the patient is at rest. This disease is undoubtedly closely allied to that form of rheumatism which chiefly affects the tendons and in many cases originates in those structures.

Lumbago, pleurodynia.

Torticollis.

Muscle tenderness.

Rest relieves pain.

General Comment.—In general it may be said of pain that in the vast majority of painful ailments the seat of pain represents more or less

Significance of seat.

exactly the location of the affected part, and any attempt to list the enormous variety is futile and useless. The more important referred pains are described under the diseases of the different organs. It should

Shoulder.

be remembered however that *shoulder pain* may be due to local disease of the joint, neuralgic disease of the diaphragm or flexures of the colon, angina pectoris and, on the right side especially, to inflammatory diseases of the liver, duodenal abscess, and movable kidney or gall stones.

Knee, groin, thigh.

Pain at the inside of the knee suggests hip joint disease; down the *front of the thigh*, ovarian and testicular disease or sometimes a developing femoral or inguinal hernia. Pain *radiating to the fold of the groin* or to the testicle is common in renal colic. Headaches have already been discussed and *the author is not convinced that they conform to any arbitrary*

Head.

classification according to location, though such has been attempted.

Lower spine.

Sacral and mid-lumbar pain is frequently of uterine origin and movable kidney often produces a painful area over the sacro-iliac joint. *Neuralgic coccygeal pain* indicates coccygodynia but equally sharp pain may

Scapula, abdomen.

accompany rectal fissure. *Pain under the scapula* most commonly indicates hepatic lesions if on the right side, splenic if on the left. *Lower abdominal-quadrant pain* suggests colitis, sigmoid irritation, appendicitis, ovaritis or varicocele.

Pubic pain is chiefly attributable to the pelvic organs and cystitis. Pleurisy and its referred pains are discussed elsewhere. *Epigastric pain* covers duodenal ulcer, appendicitis, gastric disease, (neuroses, displacement), pancreatic and vertebral diseases, pleurisy,

Epigastrim and waist line.

pneumonia, etc. *The girdle sensation* conforms to the waist line and represents constriction rather than pain. It points to injuries or tumors of the spinal cord and its meninges, chronic myelitis and locomotor ataxia. In the back *interscapular pain* is often troublesome and nearly

Interscapular and mid-dorsal.

always indicates mere indigestion or flatulent distension through aneurism, gastric ulcer and caries must not be forgotten. *Median pain at the shoulder level* may also occur in aneurism, pericarditis and diaphragmatic irritation. Ulcer of the stomach may produce *median pain lower down* with tenderness to the left of the spine over the 10th and 11th interspaces. A loaded colon, spastic constipation or mucous colitis may pro-

Lower lumbar.

duce distress in the same region. Aside from these factors *backache*, usually low down, may indicate neurasthenia, pelvic disease, excessive fatigue, spinal caries, renal disease (loin weariness is especially common),

Heel.

excessive venery and various other conditions. *Pain in the heel* may be due to sciatica, gout, ovarian and testicular lesions, intra-abdominal growth or prostatic disease. *General aching* is a common symptom

of nervous and muscular debility or fatigue, usually precedes the onset of acute infectious diseases, and is common in chronic rheumatism, trichiniasis, scurvy, locomotor ataxia, gastro-intestinal or hepatic diseases and anæmia. General.

TENDERNESS.—In general, *tenderness is associated with pain and conforms in position to the organ affected.* Only a few important variations will be noted.

The Head.—A tender *scalp* suggests syphilis, hysteria, rheumatism, neuralgia and migraine. Tenderness of the *malar bone*—neuralgia or antrum disease; of the mastoid process a mastoiditis. *Tenderness at the back of the neck*, caries and neurasthenia; *linear or interrupted spinal tenderness*, neurasthenia, rheumatism, caries, periostitis, actual spinal disease and hysteria. *Lumbar tenderness* is common in lumbago and inflammatory disease of related intra-abdominal structures; *dorsal tenderness* in advanced thoracic aneurism or posterior, mediastinal tumors of any kind. *A tender sternum or ribs* suggests ostitis or periostitis; *abdominal tenderness* is related to all acute and many chronic diseases of the contained viscera and if superficial suggests neuralgia, referred pleuritic pain or hysteria. *Hypogastric tenderness* is usually directly related to acute or chronic inflammation of the underlying structures. *The tenderness of sciatica* like its pain occurs chiefly at the sciatic notch, the middle of the thigh and knee, the ankle and heel. *Joint tenderness* is often puzzling and suggests rheumatism, arthritis deformans, synovitis, gout, gonorrhœa, tuberculosis, sepsis, hysteria or simple sprain according to the conditions developed by the case history. Frequently syphilitic.

PERVERSIONS OF SENSATION.—Paræsthesias.—The disturbances of sensation so denominated may be wholly unrelated to organic disease of the brain or cord Amongst the more important are (1). **Sensations of heat and cold.**—These suggest neurasthenia, malnutrition and anæmia, as well as hysteria, lateral sclerosis, syringomyelia and locomotor ataxia. In profound toxæmias such as alcoholism or in actual disease of the cord they are usually combined with other paræsthesias. The distribution may be general or local, indefinite, or exactly like actual contact with a cold object.

(2). **Formication and Itching.**—Itching may be pronounced in hysteria, neurasthenia, chronic alcoholism or gout, lead poisoning and various diseases of the cord. It is almost invariable in jaundice and one of the signs indicating the taking of morphine (nose rubbing). Both itching and formication may antedate or follow an apoplexy and the latter is a common symptom both as a result of actual irritation. Morphinism.

Cocainism.

diseases of the brain and cord, diabetes, cocainism and pelvic disturbances. (3). **Numbness and Tingling.**—Often associated with decided burning sensation these symptoms are especially common in connection with neuritis or pressure irritation of nerves and such drugs as aconite, in addition to the causes of formication and itching before mentioned.

Precordial.

(4). **Oppression.**—Precordial oppression is common in mediastinal tumors and cardiac disease, as well as in certain pulmonary lesions. In hæmoptysis it may localize the process and either precede or accom-

Epigastric.

pany the hemorrhage. Epigastric oppression may also be present in disease, but is more common in the functional disorders or in hæmatemesis. *Head constriction* or pressure is almost invariable in hysteria or neuras-

Cephalic.

thenia and pelvic oppression or "bearing down", is usually due to

Pelvic.

actual pelvic disease or in the male to diseases of the bladder. (5) **Faintness and Sinking Sensations.**—In angina pectoris these form

Serious.

with severe pain an agonizing and terrifying condition. Lesser degrees are common in gastro-intestinal ailments (chiefly functional), in all

Trivial.

of the cardiac diseases, in neurasthenia, hysteria and especially in hypochondriasis. *Syncope* or faintness may be purely a nervous manifestation, sometimes related to peculiar sights, sounds or violent emotion, or it may suggest actual disease of the heart or blood vessels, follow the aspiration of ascites or pleural exudates, slight hemorrhage or surgical operation, or result from pain, exhaustion, heat or the over administration of certain drugs. (6). **Heat and Morbid Flushing.**—Fatigue, nervous exhaustion, hysteria, pelvic disease and the meno-

Usually functional.

pause are the most frequent causes of flushing (hot flashes) and in certain individuals causeless blushing is a troublesome phenomenon. The sensation of epigastric heat known as pyrosis is frequently associated with a sense of excessive fullness as distinct from the pressure sensation previously described. It is most common in the functional disturbances of the stomach and in flatulence. (7). **Subjective Weakness.**—In hysteria and neurasthenia or in mere temporary brain fag the patient may experience a sense of positive physical fatigue, or, in the former,

Usually neurasthenic.

pseudo-paralytic symptoms either transient or persistent. In neurasthenia the "exhaustion' may entirely disappear under active and strenuous exertion or be forgotten in the interest excited by a good play or a good book. It is frequently due also to the overuse of tea, coffee and tobacco, or insufficient sleep. (8). **Globus Hystericus.**—

Hysteria.

Is a sensation of obstruction, constriction, pressure, or tickling referred to the throat, is characteristic of hysteria and *may* be associated with troublesome spasm.

INSOMNIA.—This troublesome symptom may take the form of inability to sleep, disturbed sleep, starting during sleep, early waking or prolonged wakeful periods. Its causes are too varied to permit full discussion here, varying as they do from mere temporary nervous excitement to actual organic disease of the brain. Inquiry should always be directed to the condition of the stomach and bowels, the amount of mental work done, amount of tea, coffee, tobacco and alcohol consumed, amount of exercise taken, the presence of any unusual sources of worry or mental strain, excessive study at night and, the age of the patient. Old people are as a rule, early wakers and relatively light sleepers, the conditions of childhood being reversed. Frequently a slight modification in the diet, hours of meals, relation of exercise to the meals, the cutting off of excesses of any kind or the mere admission of fresh air at night to the sleeping room will put an end to the attacks. If neurasthenia be the cause nothing short of a prolonged rest or recreation cure is of any use. Occasionally climatic change alone will produce sleep and permanently correct the disturbance. *In acute diseases the amount of sleep is important and should be carefully noted.*

Necessary data.

VERTIGO.—This is in most instances a trivial and transitory symptom depending upon disturbances of digestion or neurasthenia, but nevertheless should always receive careful attention. In the young the causes are usually trivial and removable. In the middle aged and old it is much more significant. The lines of investigation indicated are as follows:—The *Existence* of *Neurasthenia.* This condition covers by far the greater number of cases, associated as it is with digestive trouble, eye strain, hysteria, lithæmia, use of narcotics or stimulants, etc. *Ménière's Disease.* A condition supposed to be due to disease of the labyrinth or semi-circular canals and characterized by the association of vertigo with tinnitus aurium and a tendency to fall to the right or left. See p. 565. *Arterio-sclerosis* and *Cardiac Disease.* This vertigo covers the greater number of cases observed in *elderly people* and is important as related to threatened apoplexy, to aneurism, aortic regurgitation, other cardiac lesions or cerebral disturbances due to other causes. *Sunstroke or Heatstroke.* Sometimes leave behind a marked vertiginous tendency. *Eye Strain.* Errors of refraction and ocular insufficiencies should be carefully investigated. *Epilepsy.* Vertigo may be either an aural manifestation of major epilepsy, or represent an attack of petit mal. **The Habits.**—In regard to the use of tea, coffee, tobacco, alcoholics and the question of sexual excess these should be carefully investigated. *Brain tumors, cerebral syphilis, loco-*

Usually trivial.

Age.

Peculiar syndrome.

Common cause, in the old.

Excesses.

motor ataxia and *Bright's disease* are some of the many conditions of which vertigo is a symptom.

Subjective vs. Objective.

Varieties of Vertigo.—Vertigo is usually sudden in onset and intermittent or periodic in type. It may take the form of a subjective sensation of whirling or falling which persists with the eyes closed, or it may occur only when looking at moving objects, riding, driving, swaying, or swinging. It usually disappears or is greatly relieved when the patient is lying down, but in some instances, and notably in *its arterio-sclerotic form it is increased by lying down.* Very often a sudden change from the recumbent to the erect posture produces it, and it may be limited to the early morning hours.

Effect of posture.

Scope of inquiry.

Points to be Remembered.—*Persistent vertigo not distinctly associated with digestive disturbances or neurasthenia demands a thorough investigation of the heart and blood vessels, the urine, the reflexes, a history of syphilis, of possible epilepsy and the symptoms of brain tumor including an examination of the fundus oculi.**

Definition.

DYSPNŒA.—*The term "dyspnœa" should be limited to those cases of difficult or greatly accelerated respiration in which there is insufficient oxidation of the blood* due either to actual obstruction to the free ingress or egress of air, a diminished pulmonary area, or impairment of the chemical exchanges of the blood. *The term "accelerated breathing" should cover other varieties.* Dyspnœa may be purely subjective, but is usually both subjective and objective, and is generally associated with varying degrees of cyanosis. Any condition preventing the free entrance of air into the lungs will cause it, hence it accompanies severe quinsy and narrowing or stenosis of the glottis, trachea or bronchi, whether the cause be direct obstruction as from tumor or foreign bodies; inflammation, as in the case of laryngeal diphtheria and broncho- or lobar pneumonia; or spasm, as in croup or asthma. Other cases are referable to circulatory disturbances, especially those of the congenital type, or incompensated mitral and tricuspid lesions, in which cases the condition is largely one of pulmonary stasis, the aeration areas being normal but the blood current obstructed and its chemical exchanges diminished. In other instances both factors are concerned, as for example in emphy-

Causes.

Subjective vs. Objective.

Obstructive.

Circulatory.

* In a recent case a persistent vertigo was only explainable through the history of a severe accident, probably associated with unrecognized fracture at the base of the skull, an incident almost forgotten by the patient and not developed by the first examination. In another Ménière's syndrome was present in great part but associated with marked middle ear disturbance, valvular leakage and profound neurasthenia, and largely disappeared after a few weeks.

sema where there is both pulmonary stasis and insufficient air exchange, and to a less degree the same conditions prevail in pulmonary fibrosis, the pressure of pleural and pericardial effusion and advanced pulmonary tuberculosis. In the last instance the amount of dyspnœa is often strikingly disproportionate to the area of lung involved though usually marked upon exertion. In severe anæmias the impaired hæmoglobin content alone or a combination of circulatory and hæmic insufficiency is accountable.

Pressure. Phthisis. Anæmia.

Stertorous Respiration.—In profound coma, extensive post nasal adenoids, or greatly enlarged tonsils as well as in ordinary deep sleep in certain individuals there is a snoring respiration which is not however a true dyspnœa in most instances.

Stridor.—Stridulous breathing is invariably of laryngeal origin and usually due to spasm, paralysis, edema, foreign bodies, membrane, tumor or severe inflammation of the glottis. In certain cases it represents pressure and resulting spasm or paralysis of the laryngeal musculature as in aneurism, enlarged bronchial glands, massive pericardial effusions, etc. It is typified by the "crowing" of a croupy child, and invariably represents true dyspnœa of the obstructive or stenotic type. Its diagnostic features are stridor and a pronounced vertical movement of the larynx, the descent in inspiration being oftentimes so great as to embarrass the surgeon in tracheotomy. *The breathing is slow in obstructive dyspnœa so long as respiratory power remains good.*

Usually glottic stenosis. Croup. Important sign. Rhythm.

Air Hunger.—This peculiar form of dyspnœa is best illustrated in diabetic coma and best described by the name given.

Diabetes.

Dyspnœa on Exertion.—This accompanies severe chronic bronchitis, emphysema, early tuberculosis, pleural effusion of the latent type, anæmias, exhausting diseases or debility, obesity and imperfectly compensated cardiac lesions.

Significance.

Persistent Dyspnœa.—This indicates severer grades of the conditions mentioned in the preceding paragraph, stenosis of the air passages or certain profound toxæmias. It is particularly marked in obstructive lesions, terminal cardiac incompensation and advanced emphysema.

Paroxysmal Dyspnœa.—The commonest forms are croup and true asthma, the one being an inspiratory dyspnœa, the other predominatingly expiratory. Spasm of the glottis may be associated with hysteria or organic nervous disease, but true bronchial asthma may be perfectly simulated by dyspnœic paroxysms sometimes observed in uræmia and by a paroxysmal dyspnœa associated with cardiac lesions for which

Croup. asthma. Uræmia.

Cardiac.

the term *cardiac asthma* should be reserved. The ordinary form associated with cardiac disease being non-paroxysmal and usually extreme only under exertion, or the pressure of secondary transudates.

Glottic tumors.

Pedunculated tumors below the glottis may cause violent and even fatal paroxysmal dyspnœa.

Associated Changes in the Chest Outline.—Certain permanent changes such as the forced inspiration type of emphysema, the *unilateral enlargement* of chronic pleural effusion or tumor and the *unilateral retraction* of fibroid phthisis and pleural adhesion need no further description here.

Asthma.

In asthma, the lower diameters of the thorax are especially increased during the paroxysm because of the expiratory type of the spasmodic dyspnœa.

Broncho-pneumonia.

In broncho-pneumonia and other forms of obstructive dyspnœa a marked inspiratory narrowing and recession of the interspaces occurs because of the inspiratory negative pressure and the direct drag of the diaphragm. This need not be confused with the slighter contraction sometimes observed in chronic emphysema, enteroptosis and even in normal individuals.

Orthopnœa.—This term refers especially to the sitting attitude necessitated by extreme dyspnœa in the recumbent posture, and is most commonly noted in cardiac incompensation, or conditions producing pressure within the mediastinum.

Usually cardiac or mediastinal.

In some instances the patient must not only sit up, but is more comfortable leaning forward.*

Subjective Dyspnœa.—The mere *sensation* of dyspnœa is practically limited to hysteria or neurasthenia, and even though orthopnea be present there is no associated cyanosis.

A neurosis.

VARIATIONS IN RESPIRATORY RHYTHM.—Mere irregularity is common in children whether awake or asleep. It also appears as an *ominous sign* in massive cerebral apoplexies, brain tumor, meningitis, shock and collapse, and not infrequently as a symptom of chorea.

Jerky Respiration.—This may be inspiratory as in hydrophobia, asthma or hysteria or expiratory and often grunting in intercostal neuralgia, acute pleurisy, rib fractures, stab wounds, renal and gall stone colic, *or, in the presence of any paroxysmal pain especially of the abdomen.*

Grunting.

Wavy.

Wavy inspiration characterized by an undulatory movement of the chest may be noted in severe typhoid, pneumonia and other conditions associated with great prostration.

Biot's Respiration.—This term applies to respiration interrupted

* It has been stated that this latter position is pathognomonic of acute aortitis, but in the author's personal experience it is a common symptom in mediastinal new growths.

regularly or irregularly by apnœic intervals of varying length. In a moderate form it may be encountered in pneumonia, but is ordinarily a precursor of death in various affections.

Cheyne-Stokes Breathing.—This covers also irregular breathing interrupted by apnœic intervals but characterized by the fact that, following a pause, the respiration recommences as shallow slow breathing, increasing in depth and frequency until a maximum is reached, when it subsides in the inverse order. It ordinarily accompanies the coma of organic disease of the brain, apoplexy, uræmia, myocardial degeneration and meningitis. It may also be observed in diabetes and various acute infections, such as cerebro-spinal fever, septicæmia, typhoid, pneumonia and the exanthemata. In normal children it may occur during sleep, in the adult suffering from chronic disease of some of the types mentioned it may be present both waking and sleeping for long periods, and in a case reported by Osler was so severe and troublesome as to interfere with eating. It is usually a precursor of death nevertheless. Traube's theory of lowered irritability of the respiratory centre if combined with the fatigue hypothesis of Rosenbach would adequately cover the phenomenon.

Peculiar rhythm.

Serious sign.

Ambulatory cases.

Increased Frequency.—Mere fever increases frequency as do most dyspnœas not stenotic in type.

Hiccough.—This sudden spasmodic contraction of the diaphragm often temporary and negligible may become one of the most serious complications of acute or chronic disease. The attacks, ordinarily short, may be prolonged for days or weeks and rapidly exhaust the patient. It may be due (a) to organic or functional disease of the nervous system, (b) of the abdominal organs, (c) constitutional diseases, (d) miscellaneous causes such as severe acute infections, the typhoid state, alcoholism, pneumonia, pulmonary gangrene, chronic heart disease, pregnancy, etc. It would be useless to give in detail the various conditions with which it is associated as *it may occur in any exhausting disease, acute or chronic.* Occurring with inflammation of the diaphragm whether from the pulmonary or peritoneal side it constitutes an agonizing complication.

Often serious.

Etiology.

SHOCK AND COLLAPSE.—*The symptoms* are:—lowered temperature, cold wet surface, weak thready pulse, great prostration, pallor, Hippocratic countenance or an expression of great anxiety, combined with extraordinary mental clearness. Previously existing pain and distress are often greatly relieved and occasionally misinterpreted by both patient and physician. It is commonly associated in medicine with

Facies.

Misleading signs.

Usual causes.

those intra-abdominal diseases which have a surgical side, for example, perforating appendiceal, gastric, or duodenal ulcer, hepatic abscess and acute hemorrhagic or suppurative pancreatitis. Amongst other causes are strangulated hernia, severe accidents, particularly those attended by crushing violence, and, in its lesser degrees, even in the severe acute infections, renal and hepatic colic, and in conditions like angina pectoris associated with *extreme* pain. Its symptoms should be clearly borne in mind and its major form strongly suggests a dangerous and probably terminal complication of some existing ailment.

Shock and collapse.

Concealed Hemorrhage.—An extensive open hemorrhage almost invariably greatly alarms and excites its victim; internal and concealed hemorrhage presents symptoms of shock and collapse together with a certain curious restlessness and oftentimes yawning, nausea and air hunger. In any case of shock or collapse the stools should be carefully examined for blood and in married women the possibility of ectopic gestation cannot be ignored. The clear mind of the earlier stage may yield to delirium and occasionally there are convulsions.

Sources of Concealed Hemorrhage.—Amongst the most common are hæmothorax, aneurismal rupture, duodenal ulcer, typhoid, gastric ulcer, ectopic gestation or pelvic hematocele from any cause, traumatism, the hemorrhagic diathesis and very rarely in pulmonary tuberculosis a large hemorrhage into an old cavity may for a time be concealed.*

DISEASES OF THE THORACIC VISCERA.

THE TOPOGRAPHICAL ANATOMY OF THE CHEST.—The Regional Divisions.—*The angulus Ludovici* or angle of Louis, a ridge marking the junction of the manubrium with the gladiolus of the sternum, indicates the lower border of the aortic arch, the bifurcation of the trachea, the junction of the borders of the right and left lung, and the second rib. *The nipple* indicates, ordinarily, the fourth interspace but is subject to marked variation in position, particularly in the female. The lower border of the *pectoralis major* should correspond to the upper border of the sixth rib, and, posteriorly, the upper portion of *the scapula* corresponds to the second rib; its inferior angle to the seventh or its interspace; the root of its spine to the third rib and the right inter-lobar fissure, a region most important in connection with invasion by the tubercle bacillus. The modern regional divisions

The sternal angle.

The nipple.

The great pectoral.

The scapula.

Interlobar fissure.

* For Hæmoptysis see p. 156. Hæmatemesis p. 250. Epistaxis p. 118.

and orientation lines necessary to verbal or written description, are clearly shown by the plates.

The Thoracic Viscera.—The heart with its great vessels, the lungs and their primary bronchi, are *contained* viscera, the liver, spleen and

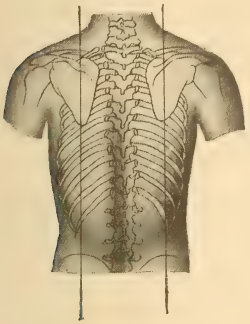


Fig. 12.—Regional Divisions of the Chest, (Posterior surface-verticals). *a, a.* Scapular lines.

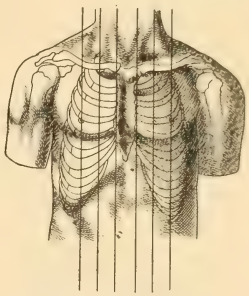


Fig. 13.—Regional Divisions of the Chest, (Anterior Surface-verticals. The various anatomic divisions are plainly indicated. *a, a.* Sternal lines. *b, b.* Parasternal lines. *c, c.* Mammillary or nipple lines.

"Contained" vs. "Sheltered."

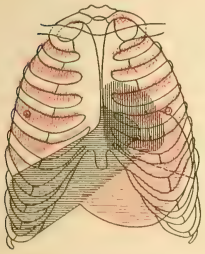


Fig. 14.—Percussion Areas, Normal Chest, (Anterior Surface.) Lungs—red. Liver—horizontal black lines. Relative cardiac dulness—vertical black lines. Absolute cardiac dulness—cross-hatching. Stomach tympany—oblique red lines.

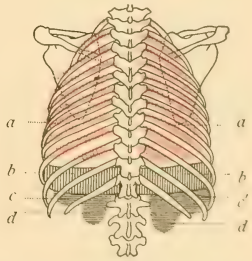


Fig. 15.—Percussion Areas, (Normal Chest, Posterior Surface). *a, a.* Lungs. *b, b.* Pleural space. *c.* Spleen, *c'.* Liver. *d, d.* Kidneys. Area *b, b* yields percussion dulness from spleen, kidneys, and liver, unless lungs are distended.

kidneys, and even a part of the stomach are *sheltered* viscera, though actually sub-diaphragmatic and abdominal.

The Lungs.—The lungs occupy nearly all the upper chest, their *apices* extending to the level of the seventh cervical spine behind and

Lung boundaries.

an inch to an inch and a half above the clavicle in front. The right is slightly higher than the left, and their *resonance* may be elicited over the whole of the supraclavicular and suprascapular regions. Their anterior borders meet at the sternal angle and, in contact, pass vertically downward to the level of the fourth cartilage; thence the left lung passes

Superficial cardiac area.

outward, forming the left border of the superficial cardiac area and the right continues downward to the sixth cartilage and in the midclavicular line both lower borders are represented by that rib. In mid-axilla they cut the eighth; in the scapular line, the ninth, and near the spine reach the level of the tenth spinous process. *From the midclavicular line outward the lower border is practically horizontal.* Below is an important area including the liver, spleen, stomach and kidneys. *The liver* lying beneath and adapting itself to the dome-like surface of the diaphragm, rises into the thorax to a much greater degree than is shown

Liver dulness.

readily by percussion, but *absolute liver dulness* begins at the lower border of the lung and extends downward to the costal margin.*

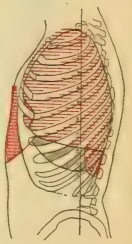


Fig. 16.

Traube's space. Bounded by the lung, spleen and liver and the costal margin. Shows region of pleural sinus in which movable dulness may appear in left-sided pleural effusion.

Traube's Semi-lunar Space.—This is included between the lower border of the left lung, the spleen, the inferior costal margin, and the left lobe of the liver, and is normally hyperresonant because of the

Diagnostic uses.

underlying stomach. *The pleuræ* extend much lower than the lung margin, being two inches inferior in the mammary line; reaching a maximum of four inches in mid-axilla, and one and one-half inches in

Pleural sinuses.

the scapular line. It will thus be readily seen that

Shifting dulness.

any small effusion of fluid into the left plural sac will, if free, produce an area of movable or shifting dulness in Traube's space, and that any increase in the size of the left lobe of the liver or of the spleen may reduce its lateral dimensions.

Delineation.

The Lobes of the Lung.—A line drawn from the second dorsal spine under the armpit to the middle of the sixth costal cartilage, divides the lung into its upper and lower lobes. On the right side a second line drawn from the middle of the first line to the fourth chondro-sternal

Relation to surface.

joint marks the upper boundary of the middle lobe. *The front of the chest, therefore, largely represents the upper, the back the lower lobe,* the apex being accessible both anteriorly and posteriorly, and the middle lobe on the right occupying a portion of the axilla and the anterior surface.

* For a description of the hepatic and splenic areas see p. 225.

The Heart.—The heart lies in the mediastinum between the lungs and presents:—(a). *A base*, at the level of the upper border of the third costal cartilages. (b). *A right border*, curving from the right base downward to the sixth chondro-sternal articulation and attaining a distance of one or one and one-half inches from the right sternal border under the fourth cartilage. (c). *A left border*, which curves outward almost to the midclavicular line, and then inward to a point one inch within that line in the fifth interspace. (d). *A lower border* connecting the lower extremities of the lines representing the right and left borders.

Boundaries.

The Aorta.—From the base, the aorta sweeps upward and to the right, its right border projecting slightly beyond the sternal margin, it passing backward and toward the left in such a manner *as to leave the manubrium resonant under normal conditions.*

Boundaries.

Manubrium percussion note.

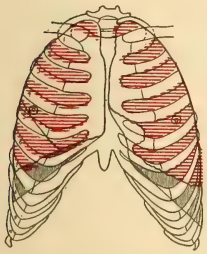


Fig. 17.—Lung Boundaries. (Anterior Surface.) (Modified Pansch-Fowler.)

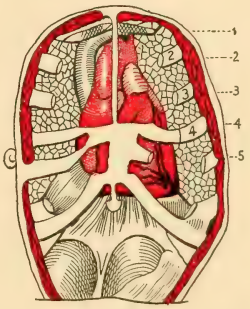


Fig. 18.—Relation of Heart and Great Vessels to Chest-wall. The lungs are pulled aside.—(After Sibson.)

Mobility of the Heart.—Suspended within the pericardial sac by the great vessels at its base, *the heart is movable and markedly displaceable* under certain pathologic conditions. *Anteriorly it presents its right chambers, chiefly the right ventricle, the left being normally represented by a mere strip of heart muscle along the left border.* The notched border of the left lung leaves a portion of the right ventricle uncovered and in such close proximity to the chest as to yield decided percussion dulness over *a somewhat triangular space having its base at the left sternal margin from the fourth to the sixth chondro-sternal articulation and its apex at or just within the apex-beat the triangle being completed by lines connecting these points.* (See fig. 14.)

Anterior aspect.

"Superficial" cardiac area.

Area of Relative Dulness.—It is so difficult to accurately outline

Practical outline.

the normal heart that many modern diagnosticians use two arbitrary percussion areas as representing the normal and serving to determine and measure any change in the boundaries of the enveloping lung or in the size of the heart. (See fig. 14.)

Apex-beat.—The apex-beat or visible and palpable heart impulse is normally found in the fifth interspace, an inch within the midclavicular line.

Anatomical position unimportant.

The Heart Valves.—The mitral, tricuspid, aortic and pulmonary valves are anatomically located within so small a space that a large stethoscope bell will nearly cover them all, but *their exact position is of little importance in clinical work.*

The Clinical Valvular Areas.—The points arbitrarily fixed for auscultation are: (a). The *mitral area* which corresponds to the heart apex. (b). The *tricuspid area* corresponding to the lower half of the sternum. (c). The *pulmonary area* corresponding to the second left intercostal space at left sternal edge. (d). The *aortic area* corresponding to the second right intercostal space at right edge of sternum.

Methods employed.

THE METHODS EMPLOYED IN THE DIAGNOSIS OF PULMONARY AND CARDIAC DISEASE.

The elicitation and interpretation of the physical signs of disease depend upon (a). *Inspection.* (b). *Palpation.* (c). *Percussion.* (d). *Auscultation.* (e). *Auscultatory percussion.* (f). *Fluoroscopy and X-Ray photography.** To a less degree one invokes certain auxiliary methods depending upon special procedures or specially devised instruments.

Know the normal.

Prerequisites of Good Work.—Every student and physician should thoroughly familiarize himself with the much neglected physical signs of *health* and the nature and extent of individual variation.

Thoroughness.—Superficial examinations are fatal to reputation and prestige. *True economy of time depends upon a correct and systematic technique and method, quick perception, and concentration.* As one avoids asking the same question twice, so should he try to fix the physical signs firmly in his memory, and the well trained man will make a complete and thorough examination and draw his conclusions, while the bewildered blunderer is still skimming the surface.

Avoid repetition.

Exceptional talent unnecessary.

Essentials.—A knowledge of topographic anatomy and of the physiology and pathology of the structure under examination; a good technique and a practised hand; good eye-sight and hearing must be combined

*Mensuration is also used but is of comparatively slight value and *spirometry* and *pneumometry* are of little use.

with an accurate knowledge of the physical signs of *health and disease. Any one who combines diligence with ordinary intelligence can become a skilled diagnostician.*

Good light.

The Preparation of the Patient.—The chest surface should be properly exposed and flooded with a light having its source behind the physician who is thus aided in his search for physical signs and his study of physiognomy and the play of a patient's emotions, for being in shadow, he can avoid betraying surprise, disappointment or dismay. *Interest, hope, cheerfulness, encouragement and calm are the only expressions allowable in the office, wards, or sick room. The arrangement of the clothing* must depend somewhat upon the sex of the patient. In dealing with women or young girls it is usually possible to conduct a satisfactory examination with some thin, soft garment covering the lower portion of the chest. In the male it should always be uncovered, and so in the female if any real necessity exists, or if by greater exposure some doubt may be resolved.* *Certainly no proper examination can be made through heavy or starched clothing, crepitations in lung apices or* heart murmurs being simulated by the crackling and rubbing of superimposed material or their conduction lost from the same cause.

Physiognomy.

Exposure of women.

Exposure of men.

A common source of error.

Attitude of Patient and Physician.—Whether in or out of bed, a patient should, whenever possible, be in an easy, unconstrained position, the tissues relaxed and the shoulders square. In the examination of the lung apices especially, both face and chest should be squarely to the front and the head quiet, otherwise deceptive differences in percussion may result from muscular contraction. The physician himself, should adopt the easiest and most unconstrained position possible for both percussion and auscultation.

Changes of Position during Examination.—*Whenever practicable patients should be examined both in the recumbent and erect posture,* because of the peculiar behavior of certain cardiac murmurs referred to further on.

An imperative rule.

EXAMINATION OF THE CHEST, WITH ESPECIAL REFERENCE TO THE LUNGS AND PLEURA.

INSPECTION.—*A glance reveals the general contour, nutrition and muscular development of the chest, the symmetry of the two sides, equality of expansion, the presence of scars, skin eruptions or abnormal growths.*

The first glance.

*Such is the confidence placed in professional honor and clean mindedness that the readiness to comply with such requests is usually a measure of the woman's refinement and real modesty.

local bulging or retraction and the absence of normal, or presence of abnormal pulsations.

Negligible factors.

General Form of the Chest.—Absolute symmetry is unusual because of slight lateral curvature of the spine, right or left handedness, or the occupation of the individual, and many variations in chest outline are trivial and negligible. *A slight wasting of muscle in the region of the lung apex is often more important* than some gross deformity. The kyphotic scoliotic or scolio-kyphotic deformities need no extended discussion but the student who has seen markedly deformed persons come to autopsy will realize the importance in diagnosis and prognosis of the displacement and crowding of vital structures. *Unilateral changes* may be due to vicarious emphysema, tumors, effusions, or congenital or juvenile heart disease. *Unilateral shrinking* indicates pleuritic adhesions, cirrhosis, collapse or cancer.

Slight wasting.

Marked deformity.

Fig. 19.—1. Unilateral Retraction. 2. Spinal Curvature.—(Gee; modified.)

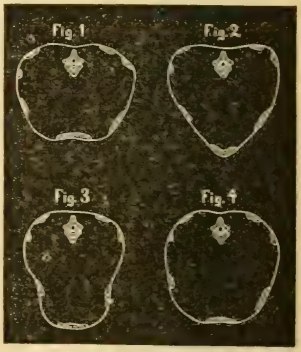
Unilateral enlargement.

Shrinkage.

General Deformities.—(Chiefly congenital). Amongst these are rachitic chest, transversely constricted chest, flattened chest, pigeon breast, "trichterbrust" (funnel breast).

The barrel chest.

Emphysema.—It should be distinctly understood that the barrel-shaped chest of emphysema is ordinarily manifest only when the compensatory curve of the spinal column is straightened out by placing the patient in a dorsal position. Such a chest often appears flattened anteriorly when the patient is erect, the shoulders being rounded and the patient slightly stooped. The *chest outline is that of permanent forced inspiration*, the epigastric angle being broad, the neck short, the stero-mastoid muscles prominent, the ribs unusually rigid and the movement more a vertical lifting than a true expansion.



Chest of forced inspiration.

Fig. 20.—1. Normal Chest. 2. Pigeon Breast. 3. Rickets. 4. Emphysema. (Gee; modified.)

The winged chest.

The "Alar" Chest.—This, also called the "pterygoid", "paralytic" or "phthisical chest," has a small anteroposterior diameter, long vertical

measurement, broad interspaces and narrow epigastric angle. The neck is long and slender and the projecting scapulæ give it the somewhat fanciful name. It does not prove a tuberculosis, but merely *suggests* the condition.

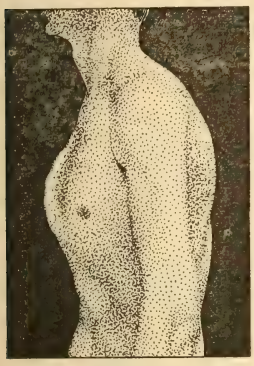


Fig. 21.
Pigeon breast.—(Rickets.)

Various Deformities.—The rachitic chest is best exemplified by the pigeon breast and the transversely constricted thorax indicates the co-incidence of deficient nutrition and some chronic obstruction to breathing in childhood, usually naso-pharyngeal adenoids. The line of retraction is the line of diaphragmatic attachment indicating its close relation to imperfect chest expansion. A beading of the ribs at the chondro-costal articulations is well known as the "rickety rosary".

Rachitic keel-shaped and constricted chests.

Rachitic rosary.

"Trichterbrust" (funnel breast) is of little importance in diagnosis, though an interesting and striking phenomenon. It is represented by a groove *deepening from above downward* and corresponding to the second portion of the sternum, the ensiform often pointing sharply forward. Usually congenital, it may be occupational in those who in early life have performed work necessitating continuous pressure over this region. The "*thorax en bateau*" observed in certain cases of syringo-myelia shows a deep median groove in the chest anteriorly.

Usually negligible.

Congenital usually.

Fig. 22.—Funnel Breast.—(Trichter brust). In this case congenital.

Localized Changes in Outline.—These are of much more significance than general deformities, and marked retraction of one side or localized retraction in any area nearly always means an old pleurisy, fibroid phthisis, past tuberculosis or injury.

Usually important.

Apex Retraction.—Symmetrical retraction of the apices due to nasal or tonsillar obstruction is common, negligible in the absence of physical signs and even in adults may promptly disappear after operation. *Unilateral* infraclavicular or suprascapular hollowing suggests existing or past disease of the lung or pleura,

Symmetrical.

Unilateral.

Insurance requirement.

Chest Measurements.—The life insurance requirements in regard to dimension and freedom of expansion are extremely simple. They demand that the circumference of the chest at the level of the armpits shall equal one-half the height of the individual in inches, and that the difference between full inspiration and forced expiration shall not be less than 2 *inches*.

An absurdity.

As a test of lung capacity this last requirement is an absurdity, for one who cannot expand under proper instruction one-tenth his chest circumference in inches can hardly be considered normal. In measuring chest

Use of tape.

expansion, one should draw the tape very *closely* at the level of the nipple, especially in fat individuals. *Abdominal measurements* should show the maximum girth with *lightly* drawn tape and should never exceed that of the chest in full inspiration, as statistics show that such

Bow windowed risks.

bow-windowed persons furnish a heavy early mortality.* *Marked unilateral variations suggest retraction due to fibroid phthisis, pleural adhesions or spinal deformity, or, bulging caused by pleural effusions and new growths.*

Costal vs. Abdominal.

Chest Movements.—Normal breathing is of two types, (1) *costal*, (2), *abdominal*. The first predominates in corset-wearing women, the second in men. *That of women is largely superior-costal, that of men inferior-costal and abdominal combined.*

Normal breathing.

In normal breathing, the ratio of respiration to pulse rate is about as 1–4, its rate is in the newborn 40–45, at the age of five 22–26, it should be symmetrical, easy and quiet, and the two sides of the chest should move *equally* and *synchronously*.

Counting respiration.

The respiration is best counted by watching the rise and fall of the epigastrium, coincident with the action of the diaphragm, or, the upper chest of women. If one ostensibly counts the pulse, keeping the eye on the proper area, neither clothing nor bed covering prevents the use of this method nor does the patient's selfconsciousness interfere. (See also Dyspnœa p.76.)

Inspiratory rhythm.

Litten's Diaphragm Phenomenon.—This is a *phantom shadow* of inspiratory rhythm passing downward from the antero-lateral aspect of the sixth rib and vanishing just above the costal margin. It corresponds to the suction exerted upon the intercostal spaces by the separation or "peeling off" of the pleural fold coincident with the descent of the diaphragm and inferior border of the lung, and its movement should

Cause.

Range.

range from 2 to 4 inches in the normal chest. *To elicit the sign*, the patient should be placed upon the back facing the light, the shoulders

*Oddly enough, *athletes* and *consumptives* usually show the highest figures in chest expansion, because both have been especially instructed and the latter practise deep breathing as a therapeutic measure.

being somewhat elevated. The observer should stand with his back to the light, five or six feet away opposite the patient's knees. The light should come from one window only, and be not too intense. It is readily seen in all normal chests not heavily overlaid by fat, but *is lost or interrupted in pleural adhesion, and absent in effusion, pneumonia of the lower lobe or tumors occupying the lower chest and greatly diminished in range in incipient or advanced tuberculosis and emphysema.* On the other hand, tumors or fluid below the diaphragm may not entirely obliterate it, and as a differential factor in the diagnosis of subphrenic abscess it has not borne out its early promise.

How affected by disease.

FLUOROSCOPIC METHODS.—As the descending diaphragmatic shadow accurately determines the relative range of excursion of the lower border of the lung, *this is one of the best methods for detecting incipient tuberculosis, a small area of infiltration often checking to a marked degree the movement of the affected lung.* The physician should familiarize himself with the range in normal breathing.

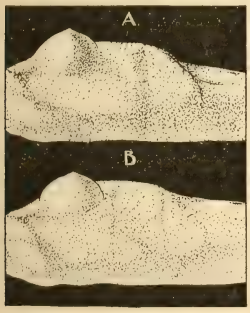


Fig. 23.—Litten's sign. (a) Reduced excursion in pulmonary tuberculosis. (b). Normal excursion.

Palpation *as applied to pulmonary disease has chiefly to do with the detection of fremitus, pulsation and lung expansion, but also determines the form, consistence, extent, mobility and sensitiveness of morbid growths, the nature of swellings, the presence of abnormal heat, the location of painful areas, the presence or absence of moisture and the quality and elasticity of the skin.*

Respiratory Movements.—In this connection, palpation confirms, corrects and amplifies the result of inspection. Expansion of the two sides should be uniform, coincident and equal in deep breathing and one notes:— (1). *Generally increased or diminished movement.* (2). *Unilaterally retarded* inspiration (inspiratory lagging). (3). *Unilaterally retarded* expiration (expiratory lagging). The hands are placed fairly upon the chest, palm down and the patient sits facing the light while the physician views the chest tangentially from behind. Two pencils, toothpicks, or matches, placed between the fingers better show the extent of movement. For the upper lobe it is well to place the thumbs in the supraclavicular space and the fingers in the infraclavicular region. *General lack of expansion may be due to deficient lung capacity, emphysema, a rigid chest wall, or a lack of skill on the part of the patient.* Many

"Lagging" and its opposite.

Technique.

Deficient expansion.

persons cannot breathe or cough to order, some do not know how to expand their lungs. *Unilateral defects of expansion* indicate a crippled lung, be the cause what it may. *Deficient upper-chest expansion in women, even if bilateral, suggests apical lesions or nasopharyngeal obstruction, for costal breathing is the normal feminine type. Superior costal breathing in men should lead one to examine the lung bases and abdomen.* Any lack of the normal inspiratory fulness in the epigastrium, unilateral or bilateral, such as is seen in *diaphragmatic paralysis* and *painful abdominal affections* should be noted. The breathing of a man who has a *painful growth of a movable subdiaphragmatic organ* is likely to be shallow and of the feminine type. In the detection of *tender areas*, the expression of the face is the safest guide. *Pleural friction and coarse rales may be palpable.*

Reversed type.

Epigastrium.

Tender areas.

Vocal Fremitus.—Vocal fremitus or the vibration of the chest caused by the production of sounds at the glottis is best detected by simultaneously placing the ulnar surfaces of both hands upon the chest at corresponding opposite points. The patient is then asked to enunciate clearly the word "nine," or "ninety-nine," the resultant vibration of the chest under the hands being noted and that of one side carefully compared with the other. It is a useful procedure alternately to raise and lower the palpating hands, thus rendering the contrast in fremitus more distinct, or apply the same hand, first to one side and then to the other. *There is normally a perceptible difference between the right and left side, that of the right being the stronger. A marked difference is a suspicious sign, especially if the fremitus of the left exceeds that of the right side, and even an equality of fremitus should suggest a special examination of the left apex.* Vocal fremitus conforms to the laws of sound conduction. In general it follows therefore—1. *That if bronchial communication exists, the louder and deeper the voice, the greater is the fremitus.* 2. *The denser the conducting material, the greater the fremitus*, e.g., *consolidated lung yields increased fremitus provided that it is in contact with, or surrounds a patent bronchus.* 3. *The transmission of the sound through a tube and into an air chamber causes conservation and reinforcement of the sound-waves and thus increases fremitus*, e.g., cavity communicating with a bronchus causes increased fremitus. 4. *The more homogeneous the conducting medium, the greater is the intensity of vibration* (fremitus). 5. *The interposition of substances of a different molecular structure between the conducting body and the palpating hand acts as a damper and interrupts the conduction of vibrations*—e.g., absence of fremitus in extensive pleural

Author's method.

Alternating palpation.

Normal predominance.

Laws of fremitus.

Voice.

Density of structure.

Reinforcement.

Structural homogeneity.

Sound dampers.

effusion, liquid or gaseous, or diminished fremitus in pleural adhesions. This may also be due in a measure to a relaxed condition of the lung.* The following points are to be borne in mind:—(a). *Markedly increased fremitus points to consolidation of lung tissue or cavity formation.* (b). *Markedly diminished fremitus indicates emphysema, pleural adhesions, pleural effusions, pulmonary edema, obstructed bronchus,* etc. It must not be forgotten that the strength and pitch of the voice and the presence of fat and muscle modify fremitus. *Also that bilateral increase or decrease in corresponding chest areas is, as a rule, of comparatively slight significance.*

Increased.

Decreased.

Pressure Palpation.—By a mere thrusting pressure of the finger tips resistance areas may be easily defined, and the method may be used as auxiliary to percussion in outlining organs, detecting exudates and *large* areas of infiltration.

Fig. 24.—Percussion of Apex. One of several faulty methods. The pleximeter finger is not flatly applied to the chest.

PERCUSSION.—The body may be directly struck with the tips of the fingers (*immediate*) or a plexímeter such as the finger, an oblong piece of *hard* rubber, or a pencil, may be struck by the finger or a percussion hammer (*mediate*). It matters little what the physician uses as a plexímeter provided he adheres to one method, *but the finger cannot be lost or left behind. The percussion stroke is best made with the middle finger, and should be given with a loose wrist as if striking a single staccato note on the piano.* It is useless to get a special plexor and plexímeter, for two pencils, a coin and a pencil, etc., etc., are quite sufficient even for the rod plexímeter percussion. Certain matters are very important:—(a). *The stroke should be equally strong on both sides.* (b). *The plexímeter finger should be placed accurately but lightly upon the chest.* (c). *Exactly the same area on each side should be alternately percussed.* (d). *No change in position that involves muscular action upon one side should be permitted.* (e). *The attention should be so concentrated upon the tone elicited as to render unnecessary a prolonged tap-*

Immediate and mediate.

Finger plexímeter.

The stroke.

Vital points in technique.

*The author is aware that the technique here recommended is not that in general use, but personal experience has convinced him that the use of the ulnar surface of the hand is more satisfactory than the employment of the palmar surface.

ping of the same region and resultant loss of time. (f). *The force of the stroke should be determined by the nature of the underlying structures.* (g). *The stroke should be perpendicular to the surface percussed.*

Seldom needed.

Strong percussion is useful when the chest wall is very fat or muscular, or when one wishes to detect some non-resonant body lying beneath one that is resonant, but should be avoided when possible as too greatly extending the area of vibration. A stroke loud enough to be heard throughout a large class room is always faulty and the term strong is relative and *does not mean a pounding stroke.**

Fig. 25.—Normal Chest. This figure shows the conditions affecting percussion of the normal chest. *a*. Variation in shape and volume of the two lungs. *b*. Modified resonance due to ribs and sternum overlying pulmonary tissue. *c*. The uselessness of percussion near the spinous processes of the vertebral column.

The best usually.

Light or moderate percussion is more generally useful and especially so when one has to deal with children, a thin wall, or with a non-resonant body overlying a resonant one.

Auscultatory Percussion.—The combination of auscultation and percussion is a valuable procedure in competent hands and especially so in determining the boundaries of thoracic or abdominal organs and the detection of cavities, or the "coin sound" of pneumo-thorax. For

Valuable.

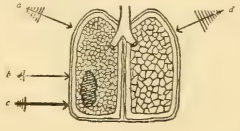


Fig. 26.—*a*. Normal. *d*. Hyperresonance (emphysema). *b*. *c*. Heavy and light percussion over consolidation.

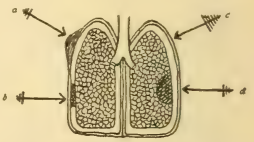
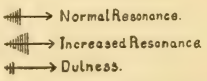


Fig. 27.—*a*. Dulness from thick wall. *b*. Pleural adhesion. *c*. Normal. *d*. New growth.



the latter it is best practised by the "rod pleximeter" method with a small coin and a lead pencil, or, in pneumo-thorax, *two coins*. The stethoscope is placed over the part to be tested or bounded and

* The nail of the plexor finger should be cut short so that only the pulp of the tip meets the pleximeter finger, and, as in golf, billiards or driving a nail, *the eye should be fixed upon the exact centre of the pleximeter* where the impact should come. The finger is by far the best pleximeter for ordinary purposes.

an assistant percusses lightly with the coin as pleximeter and pencil as plexor, gradually receding until a point is reached at which the specific note is lost or decidedly changed in pitch and quality. *Mere decrease of intensity unless abrupt is not reliable.* The heart, liver, spleen, lung apices, stomach and intestine are quite readily differentiated, and, save the two latter, accurately outlined by this method or better in the author's experience *by lightly tapping the surface with the finger tips*, i.e. combining direct percussion and auscultation. *No assistance is needed if this method be used.*

Best method.

Position of the Patient.—For percussion of the *anterior surface* the hands should hang loosely at the side; for *axillary percussion* they should be placed upon the head; for percussion of the *posterior surface* they should be lightly folded, *not* placed upon opposite shoulders.*

Position of the arms.

Percussion Sounds.—It is customary to describe the percussion notes as *resonant, hyperresonant* or *tympanitic, dull or flat.* Nothing but practice will serve to differentiate these sounds but any one may be obtained by percussing some portion of the normal body. *The typical pulmonary resonance is that of the upper axilla,* and its pitch, intensity and duration should be carefully noted. *The dull note is yielded by the liver below the lung margin* and *modified dulness just above this point,* where only the thin wedge-shaped lung border intervenes. *The flat note may be elicited by percussing the thigh or the deltoid muscle. The tympanitic note is yielded by the stomach or intestine,* as in Traube's semilunar space, and an important modification, viz.:—*dull tympany is heard over the main bronchi or upper sternum.*

Descriptive terms.

Standard notes.

Flatness.

Tympany.

Characteristics of the Percussion Sounds.—All percussion notes possess certain well-recognized characteristics and percussion itself determines resistance as felt by the pleximeter finger. *Each sound has a certain quality, intensity, pitch and duration Intensity depends upon the energy and amplitude of vibration; pitch upon the rapidity of the vibrations,* varying with the tension; *quality upon the vibrating material* and *duration upon the strength and amplitude of vibration* and the density and tension of the material. *Increased resistance goes hand in hand with dulness* and guided by this alone a deaf man might make a very fair percussor. *The more air the organ contains and the greator its deep diameter the more marked is its resonance.*

Intensity.

Pitch.

Quality.

Duration.

Resistance.

Air content.

The Normal Notes.—*It is important that the normal variations*

* The latter position necessarily involves muscular tension that interferes with the elicitation of the true note. In auscultation, however, this position is useful in determining the condition of the interlobar region.

peculiar to different areas of the chest should be held clearly in mind, and these standard notes can be learned only by painstaking practice upon a sound chest. *The apices* yield normally a resonant note, clear but not intense and tending to rise in pitch (dulness) as the pleximeter finger approaches the vertebral line posteriorly, or the trachea anteriorly. *The infraclavicular space* is typically resonant, and the pitch of the percussion note is slightly higher upon the right than upon the left side. Any tendency to approach the region of a *primary bronchus* results in a note of heightened pitch, increased resistance, and shortened duration (dulness). Below the right second rib anteriorly there is increased resonance until the fifth rib is reached when the pitch rises because of the underlying solid tissue of the liver. At the sixth rib resonance ceases and a line of absolute dulness marks the lower limit of the lung and the upper border of the uncovered surface of the liver. In the *axillary region* typical pulmonary resonance persists until the eighth rib is reached. *The cardiac area* markedly modifies the percussion note of the left chest anteriorly from the lower border of the third rib downward within the nipple line. Along the whole internal boundary of the lung anteriorly the note rises as one approaches the sternum. The clavicle is usually used as a pleximeter and directly percussed, and its centre yields a markedly resonant note. Internally and externally, pitch and resistance rise rapidly, but as a pleximeter the author believes it to be fallible and misleading.

Apices.

Infraclavicular region.

Primary bronchi.

Hepatic area.

Axillary region.

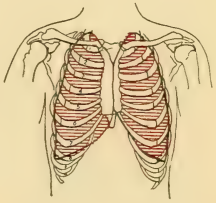


Fig. 28.—Anterior Surface. Lung Borders. Forced Inspiration.

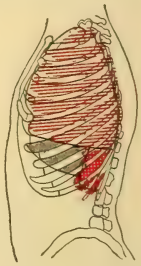


Fig. 29.—Lateral Surface. Forced Expiration.

Clavicular percussion.

Posterior Surface.—Here the height and mobility of the apices are best determined by carrying percussion upward during a forced and held *inspiration*, marking the *limit* of resonance, and repeating the procedure during forced and held *expiration*. Mere increase of intensity in the pulmonary tone in inspiration over that of expiration proves nothing.

Apex percussion.

It is the extension and diminution of the area of resonance that is important. The heavy muscles covering the back make necessary the use of greater force and the note is less clear and satisfactory than in front. This is particularly true of percussion over the scapula itself and a glance at a transverse section of a chest shows the futility of percussion near the spine. Passing downward, the superficial *liver dulness marking the lower border of the right lung* is encountered at the *ninth rib in the scapular line.*

Force required.

Liver dulness.

The Lung Borders.—*The position and mobility of the lung borders*

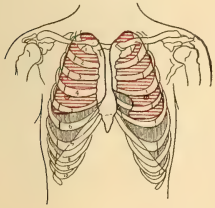


Fig. 30.—Anterior Surface. Lung Borders. Forced Expiration.

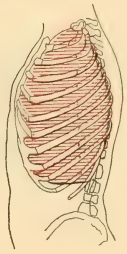


Fig. 31.—Lateral Surface. Lung Borders. Forced Inspiration.

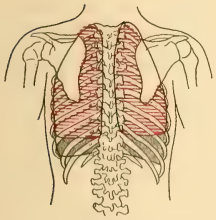


Fig. 32.—Posterior Surface. Lung Borders Quiet Breathing.

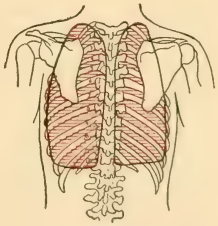


Fig. 33.—Posterior Surface. Lung Borders. Forced Inspiration.

are affected in every serious chronic disease of the lung. In tuberculosis, they show a decided lack of mobility, both at apex and base. In emphysema, primary or secondary, pulmonary engorgement and obstructive dyspnœa of any type, they are lower than normal at the base, and markedly lacking in range of movement.* In fibroid phthisis, chronic pneumonia and pleural adhesions the change in position and

Disturbing factors.

* In vicarious emphysema the loss is less marked.

movement is a striking symptom. In figures 30 to 33 the respiratory changes of the lung borders are clearly shown. *It must be borne in mind that all sound lungs cannot be held to the same limits, that equality of movement as between the two sides is the real test* and that a considerable displacement accompanies mere change of posture.

High Lung Borders.—The lung may be crowded upwards or aside by pericardial exudates, dilated heart, malignant growths, pleural exudates, meteorism, ascites or abdominal tumors.

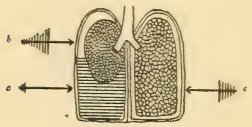


Fig. 34.—*a*. Pleural effusion. *b*. Hyperresonance above fluid exudate. *c*. Normal lung.

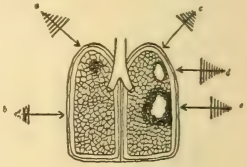
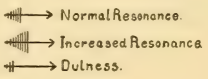


Fig. 35.—*a*. Hyperresonance over relaxed lung surrounding tubercular focus. *b. c*. Normal. *d*. Superficial cavity. *e*. Thick-walled cavity.



PERCUSSION OF THE CHEST IN DISEASE.—Hyperresonance is heard over distended, relaxed, or emphysematous lung tissue. In some cases of senile emphysema it is replaced by a note that is distinctly high-pitched and somewhat lacking in resonance.

Tympany.—The intensity and pitch of a pulmonary tympanitic note varies greatly with the size of the cavity in which it is produced, the condition of the walls and the size of the communicating bronchus. *The larger the cavity, the lower the pitch, the greater the size of the communicating opening, the higher is the pitch; and lastly, the pitch of the percussion note varies directly with the tension of the walls.*

Skodaic Resonance.—This hyperresonant or tympanitic note is heard over relaxed lung tissue, as for example, in pleuritic effusion above the level of the fluid, in the neighborhood of an advancing pneumonic consolidation, in early edema of the lungs and, to a lesser degree in certain stages of incipient apical tuberculosis. In the condition last named it may prove puzzling and lead the physician into a futile search for a lesion in the less resonant sound lung.

Relaxed tissue.

Tympanitic Note in Pneumothorax.—In open pneumothorax the percussion note is extremely drum-like, but it should not be forgotten that in many cases of closed pneumothorax the percussion sound is distinctly dull because of high tension. The tympanitic quality of any note may be lost if tension reaches a certain point.

Open vs. Closed.

Amphoric Percussion Note.—This hollow metallic note indicates a large superficial cavity freely communicating with a bronchus and so formed as to produce selective reinforcement of vibrations.

Cavity sign.

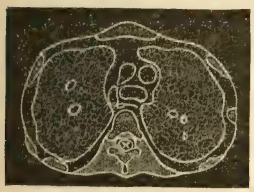


Fig. 36.—Emphysema. The distended air-cells and voluminous lung are clearly shown.

Diminished Resonance.—As resonance varies directly with the amount of air in the underlying structures accessible to a percussion stroke, deep-seated areas of consolidation covered by air-containing lung tissue yield a mixture of the vesicular and dull note. Large areas of consolidation at the surface of the lung yield a dulness exactly like that of the liver.

Modified vs. True dulness.

Flatness.—This characteristically dead or toneless note indicates dense adhesions, liquid pleural effusions or solid growths in close contact with the chest wall. The sound is of great assistance in differentiating the puzzling cases of pleural effusion in which the breath and voice sounds have almost precisely the character of those heard in pulmonary consolidation. It is by no means a difficult matter to distinguish between dulness and flatness.

Growths and liquid exudates.

Valuable sign.

Special Modifications of the Percussion Note.—The *Bruit de pot fêlé* is precisely like the "chinking" produced when the palms of the hands are placed lightly together and struck sharply against the knee and may be present normally in the thin, elastic chests of crying children. In the adult chest it can most readily be obtained in the infraclavicular region, if there be a superficial cavity with thin walls and a "slit-like" opening. The mouth should be open and the heavy percussion applied during expiration. This sign *may* be present in open pneumothorax, in the region of pneumonic areas, or above a pleural effusion.

How imitated.

Cavity.

Technique.

Friedreich's Phenomenon. The percussion note over a cavity may be higher during deep inspiration than in expiration.

Wintrich's Phenomenon.—The pitch of the percussion note over an open cavity is higher when the mouth is open.

Gerhardt's Sign.—This term is applied to the well known variation in the percussion note accompanying change of posture and is of course dependent upon the presence of movable fluid in the vomicus.

Biermer's Sign.—In pneumothorax there is a decided change in the note corresponding to changes in the patient's position.

Coin Sound.—This is described under Auscultation, on page 104.

Fundamental rule.

AUSCULTATION.—*The first law of auscultation demands that the test of symmetric breathing shall precede inference—in other words, before concluding that a departure from the type of breathing on one side represents a pathologic change, the corresponding opposite area should be investigated.* Slight symmetric departures from the normal are often transient and negligible.

Fig. 37.—Lobar Pneumonia (left); Central Pneumonia (right). The lobar consolidation on the right side would present the classical signs of complete solidification with patent bronchi. The central area of consolidation would yield no percussion signs, and be chiefly denoted by distant tubular breathing obscured by the vesicular murmur of overlying lung-cells.

Basis of Auscultatory Phenomena.—The art of auscultation rests upon the same laws of sound as underlie palpation and percussion. *The sound heard when the ear is applied to the chest is chiefly produced in the glottic chink,* but transmitted and modified by the bronchial tubes, lung tissue, and chest wall. *The nearer the ear approaches the glottis, the greater is the predominance of a tubular element in the sounds heard. If the normal pulmonary structure is replaced by consolidation, the glottic sounds are transmitted almost unmodified.* All degrees of shading may occur according to the situation of the areas of induration and their relation to the bronchi. *If the patch of thickened lung be remote from the surface, a vesicular sound due to overlying pulmonary tissue will be superadded to and modify the tubular sound.* Again, the *larger the bronchus that is in direct communication with the indurated area and the more superficial the patch, the more intense will be the sound.* Lung cavities in communication with a bronchus yield modifications of the same glottic sound, and follow the same general law. In them we have a definite air-chamber surrounded by more or less rigid walls of consolidated lung tissue; *if, then, cavities communicate with an unobstructed bronchus, and are wholly or partly empty, and if the communicating tube itself be unobstructed, the glottic sounds take on a hollow, metallic or even musical quality*

Types of breath sounds.

Modifying influences.

Broncho-vesicular.

Intensity.

Cavities.

Fig. 38.—Pleural Effusion. Especial attention should be directed to the compressed lung in the larger effusion.

Amphoric.

from the walls of the cavity and thus modified will be transmitted to the surface of the chest.

Interposition of Air or Fluid between Lung and Chest-wall.— *Any form of pleural effusion, liquid or gaseous, acts ordinarily as a damper to transmitted vibrations,* hence in any form of pleurisy with effusion, breath sounds are likely to be lost below the level of the fluid. Certain exceptions to this rule will be considered later. It will be readily understood that in the case of pneumothorax a free opening between the pleural cavity and lung may be present and that in such a case the physical signs would be those of a very large cavity. It is hardly necessary to point out the fact that *the intensity of conducted sound varies directly as the depth and intensity of the voice and inversely as the thickness of the chest-wall.*

Diminishes conduction.

Open pneumothorax.

Fat, muscle and voice strength.

Fig. 39.—Pneumothorax (left); Encysted pleurisy (right).

Mediate and Immediate Auscultation.—Auscultation may be either mediate or immediate at the pleasure of the auscultator.

Mediate.—As to stethoscopes, it matters little what one is used if the examiner is competent to interpret what he hears. The chief essentials in any stethoscope are:— (a). *That it shall clearly conduct sound in proper volume from the chest-wall to the ear.* (b). *That its chest piece or bell shall be of a size adequate for the purpose of the instrument, yet not too large or of such a form as to make it impossible to examine thin chests or those of children.* (c). *That the ear pieces are of a form and size that will completely close the external auditory canal without exerting undue pressure.* Many of the modern stethoscopes have a diaphragm and are especially useful in auscultatory percussion. The author prefers either that of Bowles or of Sansom and uses on all stethoscopes the ear pieces of the latter which are perfectly adjustable, the springs being made of malleable metal that can be bent to any degree desired. The more complicated stethoscopes have not proven useful in the author's hands, nor does he believe that it is wise to use those greatly magnifying sound. After all, there is no stethoscope as good as the unaided ear, if that has been properly trained.

Essentials.

Stethoscopes advised.

Excessive magnification useless.

Stethoscopic Pressure.—The tyro frequently inflicts much unnecessary pain by forcible stethoscopic pressure, forgetting that only such *force should be applied as will accurately adapt the whole circumference of the bell to the chest wall.* In the auscultation of heart murmurs,

Avoid over-pressure.

Effects of varying pressure.

however, it is necessary to alternately increase and diminish the pressure by which systolic murmurs are intensified and presystolic murmurs diminished.

Quiet Necessary.—A quiet place is absolutely essential to good work, for no matter what form of stethoscope be used, or how concentrated the attention of the physician, outside noise will seriously interfere with the work.

Instruction of Patient.—It may be necessary to show the person under examination just how he should breathe, and ordinarily, the lips should be closed and the respiration be deep, uniform, regular and *quiet.* Many patients will make one breath much deeper than another, or much noisier and the physician is likely to be thus misled. If nasal obstruction exists the mouth should be kept open and in any event *it is well to listen to a few inspirations* before applying the ear or stethoscope to the chest.

Quiet deep breathing.

Attitude of Patient During Auscultation.—The position of the patient during auscultation should vary with the different areas under investigation, precisely as in percussion, save that the most effective examination of the region of the posterior interlobar space requires that the hand should be carried across the chest to the opposite shoulder. The scapula is thus carried forward and outward, and by its posterior border very nearly defines the division between the upper and lower lobes.

Posterior inter-lobar region.

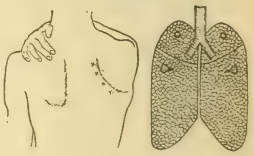


Fig. 40. Fig. 41.

Fig. 40.—An Important Area. (*After Fowler.*) Position for auscultation of interlobar region.

Fig. 41.—Selective Points in Pulmonary Tuberculosis.

Pulmonary Areas Demanding Special Attention.—Fowler, of London, has emphasized the necessity for a special examination of certain auscultation areas. He shows that tuberculosis nearly always first involves the apex; not the extreme tip, but a point nearer to the posterior than the anterior surface and somewhat external, and that it follows a definite "line of march," passing downward *and into* the upper portion of the lower lobe along the interlobar fissure. Hence the most important areas are:—(a). *Posteriorly, at a point opposite the second dorsal spine but well toward the scapula.* (b). *Anteriorly, at, or just below the middle of the clavicle.* (c). *The supra- and infrascapular space.* (d). *Along the inner border of the scapula, when the hand of that side rests upon the opposite shoulder.* (e). *The upper part of the axillary space.*

Tuberculous invasion.

"Line of march."

Regions most important.

Normal breathing.

Vesicular Breathing.—Its characteristics are a *peculiar rustling quality, low pitch, moderate intensity, and, no definite pause between inspiration and expiration.* The latter has no more than one-third the duration of the former and may be inaudible in ordinary auscultation.

Children.

Puerile or Harsh Respiration.—*Normal.*—In the chest of a child, or when listening over a lung that is performing vicarious duties, one hears the so-called puerile breathing, which is practically intensified vesicular respiration.

Pathologic.—Over the unaffected lung in cases of pleurisy with effusion or pneumonia.

Suggested ailments.

Prolonged Expiration.—Aside from the stridor of obstructive or spasmodic dyspnœa a respiratory sound possessing this as its most marked characteristic is frequently heard on auscultation, and suggests at once a difficulty in the expulsion of air from the lungs. If the expiratory note be high pitched, a small area of infiltration is indicated, if the pitch be low, then one thinks of distention or relaxation of the lung tissue, such as occurs in emphysema, chronic bronchitis, or areas immediately surrounding an incipient tuberculous deposit.

Ailments suggested

Suppressed Breathing.—Absence of the breath-sounds over any part of the lungs indicates either feeble breathing, the interposition of some substance between the lung and the chest, or an obstructed bronchus. Pleurisy with effusion and pleural growths or thickening represent the former, and the rare cases of massive pneumonia, pressure of growths or aneurism, or occlusion by foreign bodies the latter. Suppressed breathing is common at the apex in incipient phthisis and in most conditions in which there is collapse of lung tissue. In bronchopneumonia as in early apical tuberculosis, cough, or, in the child, crying, may develop well defined breathing or suggestive modifications.

Effect of cough.

Usually misleading.

Cog-wheel Breathing.—Much stress has been laid upon this as a symptom of early phthisis. It is over-rated and one should remember that mere nervousness produces marked disturbance of the heart's action, irregular and unequal inspiration, uneven muscular contraction and therefore pseudo cog-wheel breathing. *If genuine it should be heard even when the breathing is regular and deep, and the heart action neither unduly violent nor greatly accelerated.* The term "cog-wheel" is not descriptive: *the breathing is wavy, and marked by distinct breaks.* When genuine it indicates imperfect expansion of some portion of the lung.

Precautions.

Normal over 7th cervical.

Bronchial Breathing.—*Normal.*—If a stethoscope be placed over the seventh cervical spine bronchial breathing is heard. *It differs from*

Qualities.

vesicular breathing in almost every particular. Both inspiration and expiration are high pitched; between them is a distinct interval. Expiration is greatly prolonged, its intensity being equal to or even greater than that of inspiration.

Points to infiltration or compression.

Pathologic.—Such breathing indicates consolidation or compression of lung tissue in close relation to a patent bronchus. Thus it is heard in pneumonia and phthisis, and over lung compressed by mediastinal or other tumors or pericardial effusion.

Misleading variation.

For the same reason probably, the voice and breath-sounds are well conducted in some cases of effusion but in such the flat toneless percussion note suggests the correct diagnosis and the breathing is usually *distant* though tubular.

Fig. 42.—Malignant Growth (left) and Pulmonary Abscess (right). The larger mass on the left side involves a bronchus, and would yield signs of consolidation. The anterior superficial mass would present only dulness, diminished voice and breath sounds, with defective lung movement on affected side. Such abscesses as are here shown present few recognizable *physical* signs, and are often overlooked.

Normal area.

Broncho-vesicular Breathing.—*Normal.*—If the stethoscope be placed over the second intercostal space at the sternal border, or upon the upper interscapular region of the normal chest, the sound conducted to the ear is *a curious mixture of vesicular and bronchial breathing.*

Points to deep consolidation.

Pathologic.—There is no more important clinical study than this modification, as it frequently represents early tuberculosis or deep-seated central consolidation.

Glottic breathing.

Tubular Breathing.—*Normal.*—Tubular breathing is heard normally over the glottis and *differs from bronchial breathing in being more intense and possessing a peculiar whiffing quality.*

Superficial consolidation.

Pathologic.—Its significance when heard over pulmonary areas is found in its association with complete *superficial consolidation* of lung tissue. It is important that this breathing should be carefully studied, because of its intimate association with, or near likeness to cavernous and amphoric breathing.

Hollow and low pitched.

Cavernous Breathing.—The *sole important difference between cavernous and tubular breathing lies in the fact that in the former the pitch is low and the quality distinctly hollow.* For its production, a pulmonary cavity is necessary, empty or partially so, and not less than an inch (2 cm.) in diameter.

Amphoric Breathing.—*Amphoric breathing is exactly the same as cavernous, save that it possesses a metallic or musical quality,* the sound

resembling that produced by blowing across the mouth of an empty bottle. It is heard over large superficial cavities, and in open pneumothorax. *In all cases of cavity free bronchial communication is necessary to the production of the typical sound.*

Metallic and musical cavities.

Vocal Resonance.—*Normal.*—If the ear or stethoscope be applied to the axillary region of a healthy chest, and the patient slowly repeats the word "ninety-nine," the voice is heard but the words cannot be distinguished; moreover, the ear recognizes the fact that the sound is produced at a point some distance from the stethoscope. Such normal vocal resonance is associated with a vesicular murmur and a normal percussion note, but in disease we find variations to fit every variety of abnormal breath-sounds and percussion tones.

Where heard.

Characteristics.

Modified in disease.

Bronchophony (bronchial voice).—*Normal.*—Bronchophony may be heard in the normal chest over the manubrium. At this point the voice or whisper seems to be directly at the mouth of the stethoscope and the sense of remoteness, so marked in normal resonance, is altogether lost. *Pathologic.* Bronchophony is heard in varying degrees under the same conditions that produce bronchial and bronchovesicular breathing. A faint bronchophony is sometimes termed a "bronchial whisper" but should be called "distant" or "faint" to avoid confusing it with "whispered" bronchophony.

Where heard.

Peculiar features.

Indicates consolidation.

Pectoriloquy (tracheal voice).—*Normal.*—Pectoriloquy corresponds to the normal voice as heard over the trachea. It differs from bronchophony in that the words seem to be distinctly articulated and spoken directly into the ear. Whispered pectoriloquy is the term applied when the whispered voice is thus transmitted.

Where heard.

Pathologic.—It is heard over superficial cavities and consolidation.

Superficial lesions.

Increased Vocal Resonance of Heightened Pitch.—Vocal resonance may be only slightly increased, with a *rise in pitch* in cases of imperfect consolidation of lung tissue, or in cavities that are not surrounded by consolidated lung tissue.

Important variation.

Egophony.—*Pathologic.*—Egophony is a peculiar modification of the voice-sound that gives it a bleating or *distinctly nasal* character. It is most frequently heard at the upper level of pleuritic effusion, and just above the area of advancing consolidation in pneumonia. Attention is not infrequently directed to small pleural effusions, acute or subacute, by encountering egophony at the inferior angle of the scapula.

Character.

Significance.

Diminished Vocal Resonance.—The following conditions result in diminution or loss of the normal vocal resonance:

Normal. (a). Weak voice. (b). Excessively thick wall. *Patho-*

logic. (a). Emphysema or relaxed lung from whatever causes. (b). Effusion into pleural cavity (liquid or gaseous). (c). Pleural growth or thickening. (d). Massive pneumonia (large bronchi blocked). (e). Occluded bronchus due to foreign bodies or to pressure.

Pneumothorax.

Echoing Resonance.—The *coin sound* proper is a peculiar bell-like note heard over the affected side in pneumothorax when a coin is placed upon the chest and lightly struck with another. The auscultator usually applies his ear or stethoscope to the back and an assistant manipulates the coin upon the anterior surface of the chest. Echoing or amphoric resonance of the voice and heart-sounds may be heard under similar conditions.

Amphoric voice and heart sounds.

Dry and moist.

RALES.—*Râles are either dry or moist,* and a consideration of the anatomic structures of the lungs and pleura makes it easy to understand the mode of their production, their significance and the varying qualities dependent upon the condition of the lung itself and the character of the respiration. Aside from the friction râle, dry râles may be divided into two groups:—(a). *Sibilant* or whistling. (b) *Sonorous,* "at one time resembling snoring, at another the sound of a bassoon, and very frequently it is like the cooing of a turtle-dove." They are of all grades of pitch and intensity and in passing from the very feeblest sibilant to the most raucous sonorous râle, *the differences depend upon the size of the tube in which they are produced, its proximity to the surface, relation to the consolidated areas, and the depth and character of the respiration itself.* Such râles alone or in association with others are most frequently found in bronchial obstruction, bronchitis, asthma and emphysema, but persistent dry râles are common in incipient tuberculosis and may be distinctly resonant or consonating.

Sibilant and sonorous.

Musical.

Modifying factors.

Obstruction or glottic paralysis.

Stridor.—Stridor is the loud sound heard over the trachea or a primary bronchus that is obstructed by aneurism, tumor or a foreign body. A similar sound is heard in paralysis of the abductors, such as may result from pressure upon the recurrent laryngeal nerve.

Cardinal points.

Sites.

Causes.

Moist Rales.—In the consideration of moist râles *the chief points to be noted are:—their quality, size, pitch, and resonance.* A moist râle may be produced by respiration at any point from the glottis to a terminal alveolus and may be due to the serous exudate of edema, the mucous or mucopurulent secretion of bronchitis, the thick tenacious sputa of pneumonia, or the purulent fluid of a pulmonary cavity or pneumo-pyothorax. Being oftentimes associated with consolidation of all kinds, or cavity of any degree, it must vary in pitch, quality, size and intensity with its surroundings.

Crepitation.—Beginning with the smallest râle we have crepitation, a shower of tiny sounds compared to the crackling of burning salt, or better to that produced by rolling a lock of hair in the fingers, held close to the ear. Crepitation is not a liquid râle but signifies merely that air is entering into a collapsed vesicle, or, represents fine pleural friction or the separation of slightly adherent pleural surfaces.* It is one of the early signs of lobar pneumonia (*crepitatio indux*), hemorrhagic infarction and incipient tuberculosis; occurs as a precursor of resolution (*crepitatio redux*) and may normally be heard at the apex or base of the lung when the first deep inspiration is taken. Such râles are also heard persistently at the base of the lung early in pulmonary edema and hypostatic congestion. *They are heard always at the end of deep inspiration* and may be confounded by the tyro with superficial pressure crepitation produced by his own ear or stethoscope.

Smallest.

Dry râle

Cause.

Associated lesions.

When normal.

Source of error.

Crackling Rales.†—Three varieties of crackles are recognized by the diagnostician. *The fine crackling* or subcrepitant *râles* are produced in the finer bronchi, heard chiefly but not exclusively in inspiration and indicate, as a rule, commencing infiltration. Heard at the apices, they always strongly suggest an incipient tubercular process; at the base, pneumonia or congestion. They are heard early in resolving pneumonia. *Medium crackling râles* are of the same general nature as the foregoing, but coarser. They are heard over softening tuberculous areas, broncho-pneumonia, and resolving lobar pneumonia. Consonance is marked in the presence of infiltration.

Significance.

Consonance.

Large, Crackling Rales are still coarser and more distinctly fluid than the foregoing and point usually to extensive softening in a tubercular area.

Mucous Click.—This sound stands midway between the crackling and the bubbling or gurgling râles. It is a "sticky," mucous crackle usually single and commonly heard at the apices in tuberculosis.

Bubbling Rales.—These may be small, medium or large liquid râles, and are usually readily recognized. **Gurgling Rales.**—These are usually heard over large cavities during cough or in forced inspiration.

Pitch and Resonance of Rales.—*All varieties of râles have a certain pitch and degree of resonance, directly attributable to their surroundings.* One that is produced in a bronchus or cavity surrounded by

* There is certainly a pleural crepitation hardly distinguishable from alveolar crepitation and, a more pronounced pleural *crackle*.

† The author believes that the ear distinguishes easily a difference between medium or even large crackles and the similiar but more liquid "bubbling" râles. Hence the classification used.

Resonant and consonating râles.

consolidated lung tissue or atelectasis will have many of the qualities of tubular or amphoric breathing hence we speak of *resonant* râles, *echoing* râles, *consonating* râles, etc., and these may be either moist or dry. There are certain special sounds of great interest, and chief among these are "post-tussive suction," "metallic tinkling" and "succussion."

India rubber ball sound.

Water whistle sound.

Post-tussive Suction.—This interesting physical sign is pathognomonic of cavity formation and has been aptly termed "the india-rubber-ball" sound. It is supposed to be due to the expansion of a collapsed thin-walled cavity communicating with a bronchus. A similar but coarser and gurgling suction sound either inspiratory or expiratory may be heard in pneumothorax if communication with the lung exists.

Cavity or pneumothorax.

Metallic Tinkling.—This term is applied to the beautiful musical sound sometimes heard over a very large cavity with dense smooth walls or in pneumothorax. The phenomenon is really due to the production of echoes, and when low pitched is often termed amphoric tinkling.

Pneumothorax.

Hippocratic Succussion.—This is the well known swashing sound, often metallic, produced by the somewhat heroic measure of shaking the victim of a pneumohydro- or pyo-thorax.

Friction Sounds.—True friction is produced by the attrition of pleural surfaces roughened by inflammation and is usually unmistakable, often palpable. *The quality of the sound is creaking, rubbing or crackling;* it is ordinarily distinctly superficial, commonly best heard in the axillary and inframammary regions and at the anterior lung margins, is usually double in rhythm, and what is still more important, is associated in most cases with pain on deep inspiration and is unaffected by cough.* Friction râles may persist at the margins of dulness due to pleural exudate and their reappearance indicates absorption. *Pleural crepitation usually* occurs *early* in the inspiratory phase of the individual breath-sound whereas true crepitation is late but the distinction is not always possible.

Invaluable.

THE USE OF THE X-RAY IN THE EXAMINATION OF THE CHEST.—The importance of fluoroscopic and photographic methods in the diagnosis of diseases of the heart, lungs and pleura is too little appreciated by the average physician. Fluoroscopic examination has been

*It is accurately simulated by the sound heard when the palm of the hand is pressed firmly against the ear and the dorsum rubbed by the *flat* of the finger pulp.

made a part of the author's routine method for several years, and has proven itself indispensable. Either the static machine or the coil may be used, the former having as its sole advantage its applicability to therapeutic uses; the coil being much more convenient on account of its greater reliability and the short time needed to put it in operation. Full instruction regarding technique is to be found in books devoted especially to that subject, and it need only be stated that, (a). *tubes of medium resistance give the best results.* (b). *That minimum exposures should be used.* (c). *That repeated exposure at short intervals should be avoided*

Static vs. Coil.

General rules.

Beware of burns.

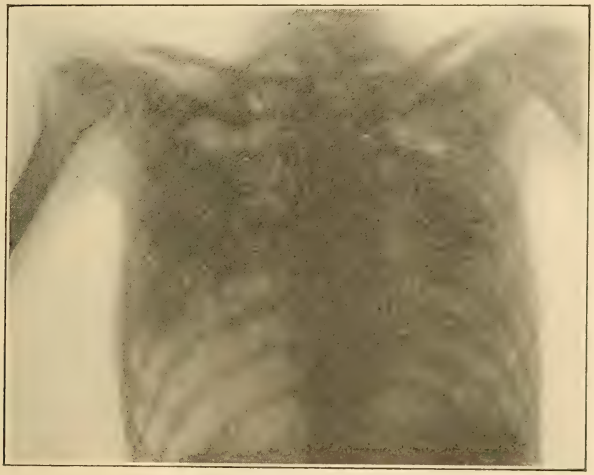


Fig. 43.—Advanced Pulmonary Tuberculosis. Multiple cavities at apices. One at right base. Infiltration marked. Compensatory emphysema with intermingled infiltration at left base.

so far as possible. (d). *That especial care should be observed in the case of patients having an irritable or diseased skin.* (e). *That the same focal distance should be observed in every case.* This is ordinarily 15 inches from the target to the surface of the body. This precaution is especially necessary for the accurate comparison of observations. (f). *A thorough familiarity with the fluoroscopic appearances of the normal chest is indispensable.* (g). *The operator must, by the use of a proper screen, avoid the exposure of his own hands, or be prepared for minor burns which may prove extremely troublesome.* (h). *A negative will often show more than the fluoroscopic picture or finished photograph.* By this method of examination areas of pneumonia, of tubercular

Use one focal distance.

Know the normal.

Value of negative.

Lesions shown.

infiltration, of pleuritic adhesion and existing pleuritic effusions may be detected. While the X-Ray is but a part of a physical examination *one is surprised to find how often ordinary physical signs have been misleading or have failed to reveal more than a part of the conditions*

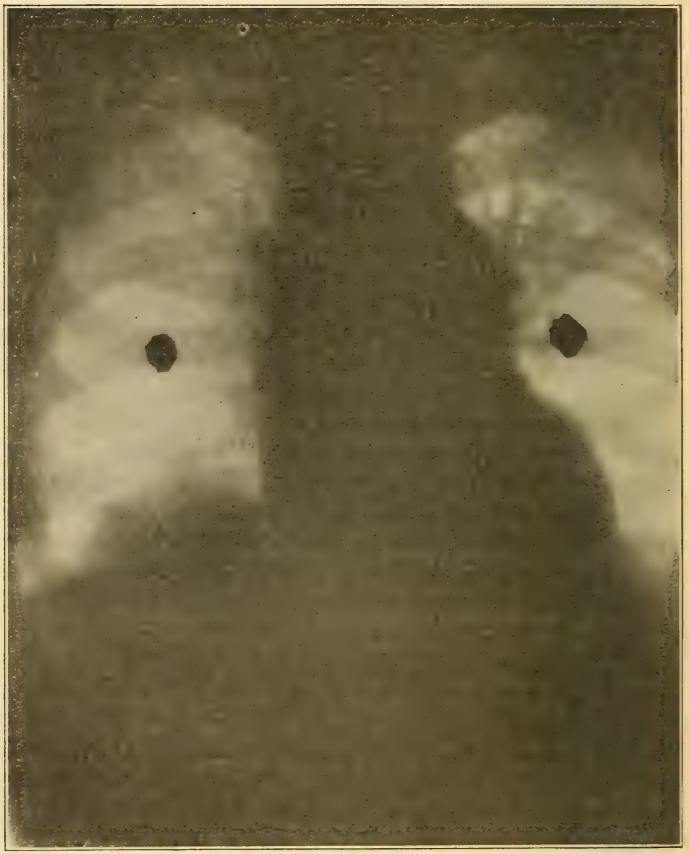


Fig. 44.—Aneurism of ascending and transverse portions of arch, classical signs absent. Death six months later from rupture into pericardium.

Often superior to physical signs.

actually existing. Often an apparently slight tuberculous infiltration is found to have involved the major part of the lung, an apparently small heart proves a dilated one, and quite often one finds a

seemingly unilateral involvement bilateral. An unsuspected cavity may show plainly as a bright reflex. In diseases of the heart the X-Ray methods are invaluable, for not only is the cardiac outline clearly and accurately defined, but the early diagnosis of aneurism or mediastinal growths is made possible. Indeed, *it is the only method by which a positive diagnosis of aortic aneurism can be made in its early stages.* The outline of the distended aorta is plainly shown, and what is quite as important, expansile pulsation, the *sine qua non* of diagnosis is made evident.

Incipient tuberculosis.

Heart lesions.

Aneurism.

Fluoroscopic Signs and Their Significance.—*Bright light reflex in shadow areas* = cavity. *Apical shadows* = infiltration usually tuberculous. *Shadows at base* = pneumonia, congestion, adhesions or edema. *Unilateral shadow associated with rhythmic lateral displacement of the heart in deep breathing* = pleural effusion. *Same unassociated with such displacement* = old pleural adhesions, malignant disease or subdiaphragmatic abscess. *Localized shadow* may indicate abscess or malignant growth, localized tuberculosis, foreign bodies, encysted pleurisy. The *clinical symptoms and physical signs* of course indicate the acute, chronic, primary, or secondary nature of the lesion.

COUGH.—*The term cough covers single or multiple, consecutive, explosive, expiratory acts preceded by a spasmodic glottic closure.* It may arise from a multitude of causes and the irritation of widely separated and diverse regions, though commonly purposeful and intended to remove irritating material from the bronchi or throat, this being ordinarily the secretion accompanying acute or chronic disease of the air passages, or, foreign bodies. *The most sensitive regions* are the interarytenoid space, the tracheal bifurcation, pharynx, base of the tongue, nasopharynx and certain areas in the nasal passages proper. Many forms of *reflex cough* are observed and these should always receive consideration in instances of otherwise inexplicable persistent cough. Arnold's branch of the pneumo-gastric nerve is accountable for a rare cough connected with irritation of the external auditory canal.* So also chilling of the surface, pressure upon the spleen or liver, or the various diseases of the liver or gall-bladder when associated with involvement of the diaphragmatic pleura; and finally, in women, chronic disease of the pelvic organs, is an occasional cause and such a cough frequently disappears entirely after operation. Other coughs are purely neurotic, such are the obtrusive *hysterical cough* and the *barking cough*, of pu-

Definition.

Diverse causes.

Sensitive areas.

Reflex cough.

Neurotic cough.

*Such a case recently observed by the author was promptly and permanently relieved by the removal of impacted cerumen.

berty. *Dentition cough* in infants is purely reflex in character, but the so-called *stomach cough*, associated ordinarily with chronic gastritis and most frequently observed in drinkers is no doubt due to the accompanying pharyngitis and the same statement applies to the *smoker's cough* and its associated laryngitis. Many cases of *excessive irritation of sensitive turbinate areas* are observed, particularly in young people and neurotic individuals, the conditions being very similar to those associated with asthmatic paroxysms and hay fever. It should be remembered that *irritation of the lung parenchyma does not cause cough*, but unquestionably *irritation or inflammation of the pleura does*, hence *in pulmonary disease, acute or chronic, the cough is attributable either to bronchial or pleural irritation*. Again it should be said that *the assumption that any cough is reflex or neurotic should be postponed until all other channels have been thoroughly investigated.*

Stomach cough.

Smoker's cough.

Nasal cough.

Pleural cough.

Source of error.

Dry and Moist Cough.—In incipient phthisis and the early stages of bronchial inflammation or irritation from whatever cause the cough is *unproductive*, hence it is termed *dry*. It may be extremely *urgent, paroxysmal*, or *painful*. Acute bronchitis, pneumonia, whooping cough, asthma, and pleurisy furnish the best examples, and, in their early stages, practically all of the nervous, reflex, or pressure coughs are dry. *When pain is present* an effort is made to *suppress the cough* as is the case especially in lobar pneumonia and pleurisy. *Paroxysmal cough* best exemplified by whooping cough may be encountered in a large number of conditions whether due to direct irritation as in the case of a laryngeal tumor, or to reflex causes as in impacted cerumen or turgescence of the nasal passages, and in pertussis particularly this paroxysm may be associated with vomiting and even with hemorrhages under the skin and epistaxis. *A paroxysmal cough may also be associated with the accumulation of secretion* and attended by a profuse and perhaps sudden discharge, as for example, in bronchiectasis, certain pulmonary cavities or the rupture of abscesses into the bronchi. *Pressure cough* due to mediastinal tumors, pericardial effusion, etc., may be, but ordinarily is not, paroxysmal. The so-called *hacking cough* is especially common in incipient tuberculosis, but may occur in chronic catarrh of the upper air passages.

Characteristics.

Suppressed.

Paroxysmal.

Reflex.

Vomiting.

Hacking cough.

Various Types.—*Barking cough*, if *not* neurotic, is usually associated with inflammation of the glottis, a *hollow cough* with advanced tuberculosis, and *ringing, metallic or brazen cough* with mediastinal pressure from whatever cause. The author has observed as good examples in massive pericardial effusion as from its commoner cause,

Barking, hollow and brazen coughs.

aneurism. *In emphysema* the cough is peculiar and somewhat characteristic in the prolongation of the individual expirations and the manifest forcing and prolonging of the series. The *hoarse cough* of croup is well known and a *noiseless cough* may occur in certain forms of glottic paralysis or a *toneless cough* in terminal cases of pulmonary disease. Emphysema. Croup. Toneless cough.

The *inspiratory whoop* may follow various forms of paroxysmal cough but is occasional and unusual, whereas in whooping cough when developed it is persistent and practically pathognomonic. Finally, the *absence or cessation of cough, with persisting physical signs*, may indicate profound toxæmia, excessive weakness or approaching death and is seen especially in fatal pneumonias of infancy, childhood and old age. Whooping. Ominous sign.

Localized momentary protrusion normally occurs in the apices and upper intercostal spaces during cough. The condition is exaggerated in emphysema and diminished in infiltration.

SPUTUM.—The sputum proper is that secretion of the air passages obtained by "hawking" or coughing and must include nasal, pharyngeal, laryngeal, bronchial, alveolar, and a certain amount of oral secretion. All save that representing pure pus or blood is mucoid in consistence and the general term embraces all similar material derived from fistulous communication with adjacent structures, or destructive processes within the respiratory tract, i.e. perforating abscess, echinococcus cysts, cavities, etc. Sources.

One must observe (a). *Reaction.* (b). *Color and transparency.* (c). *Air content.* (d). *Consistence.* (e). *Amount.* (f). *Odor.* (g). *Albumin content.* (h). *Microscopic findings.* Data.

The reaction is alkaline save after certain decomposition processes outside the body. *Aside from misleading tints derived from food or medicines* **the color** varies from the colorless mucoid or serous sputum to that of pure blood. Slight purulent admixture (*muco-purulent sputum*) or predominance of pus (*purulent*) produces various degrees of yellow or greenish yellow. *Dust and soot* inhalation gives a dingy or grayish tint and bile pigment or the development of certain germs may produce a faint or even vivid green. *Blood* may be pure or represented only by faint pink, light brown or salmon color, iron rust shading, faint yellows and greens or the thin serous so-called "prune juice" sputum. True *icteric sputum* appears only when actual jaundice or perforating hepatic abscess is present though many blood containing specimens of unusual tint react to the test for bile pigment. Blood. Bile.

Greenish tints may occur in carcinoma, chloroma and various other conditions, and various occupational sputa are encountered as in mirror polishers, aniline dye workers and others. In

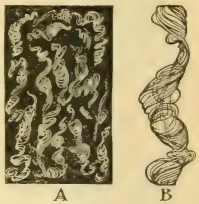
Important varieties.

disease of the mitral valve with brown sputum peculiar pigment holding cells are present; in the perforating abscess of amœbic dysentery the "*anchovy sauce*" *sputum* may be encountered, and in hysteria there is occasionally a viscid or jelly-like sputum exactly like *crushed raspberries*, which may create much unnecessary alarm.

Air Content.—Foaminess and low specific gravity, indicate the air content and all *serous sputum* is frothy. Cavity sputa appear in water as globules, flattening to coin like bodies (*nummular sputum*) as they sink to the bottom, and in general, *the greater the pus content the less the air*, and *the smaller the tubes of origin the greater the air content.* In *bronchiectasis* and *gangrene one finds three distinct layers* because of the varying specific gravity.

Striking differences.

Consistence.—A proper background is readily obtained by laying a piece of glass or a Petri dish over black paper or cloth or purchasing or making a special plate with half its surface pure black. One may find (a). "*Curschmann's spirals,*" which are not pathognomonic of asthma, but most frequent in that disease. They are refractile and visible to the naked eye, represent a bronchiolitic exudation (Sahli), are interesting, but have no great diagnostic importance. "*Dittrichs plug's*" are yellowish white and mustard seed sized, *foul smelling* aggregations of fatty acid crystals and bacteria closely resembling the tonsillar plugs of follicular tonsillitis and are found in decomposition processes (pulmonary gangrene, foul cavities, etc.). *Misleading food particles* are usually starchy and strike a blue color with Lugol's solution. *Fibrin masses* are white and extremely tenacious and are rendered clearer and bulkier by the addition of acetic acid. They vary in size from the tiniest plugs to the fibrinous casts of the bronchial tubes (croupous pneumonia, fibrinous bronchitis), or the diphtheritic membrane. *The fibrinous casts* show best if shaken up in water. Rarely and especially in old cases of tuberculosis, *calcareous plugs* often of considerable size are expectorated,* or various foreign bodies of recent or ancient introduction may appear. *Pus* content or *serum* determines the fluidity and *mucous* and *nuclein* (croupous pneumonia) the viscidity and tenacity of the sputum. All sorts of sub-



A B

Fig. 45.—Curschmann's Spirals. A. unmagnified. B. magnified.

Spirals.

Plugs.

Food particles.

Fibrin.

Fluidity vs. Viscidity.

*In a case observed sometime ago a large lung stone was ejected after a protracted and violent coughing seizure and no symptom of the old disease had been manifest for over 20 years.

stances may be introduced by fistulous communication with abscesses, echinococcus cysts, etc., etc.

Amount.—Whenever possible the 24 hours sputum should be obtained and the transparent sputum cups are far better than the metallic and paper cups so generally used. Early stages of inflammation yie'd scant secretion. The morning is the most productive period in chronic processes though in pulmonary cavity and especially bronchiectasis large amounts may be raised at irregular times, often when assuming a special posture. Advanced pulmonary tuberculosis, bronchorrhœa, bronchiectasis, perforating abscesses, pulmonary edema and resolving pneumonia furnish large amounts. Best cups.

Odor.—Ordinarily odorless when first raised it may be foul in any process attended with decomposition within the lung (bronchiectasis, certain tuberculous cavities, abscess of liver, communicating empyema) and characteristically so in gangrene.

Albumin Content.—The albumin content constitutes a rough measure of the severity of an inflammation hence some slight value may be attached to a decided percentage as differentiating simple bronchitis from pulmonary tuberculosis.* Slight value.

Microscopic Findings.—The sputum should first be examined as a flat preparation without staining and then by making a smear, drying in the air and staining for three minutes with Wright's stain, thoroughly washed, and mounted. One may find (a). *Flat pavement cells* from the mouth and pharynx. (b). *Columnar ciliated cells* from the larynx, trachea and bronchi. (c). *Mono or polynuclear oval cells* (25–50 y.) from the alveoli which may contain (1). *Irregular highly refractile masses* of myelin showing concentric layers. (2). *Blood pigment* bearing (heart disease) cells. (3). *Hematoidin crystals*. (4). *Fat*. (5). *Carbon*. (d). *Free myelin bodies*. (e). *Elastic fibres*. These if present may be found by pressing a cheesy, granular or thick portion of sputum between two glass slides and using a low power lens.† They indicate actual destruction of lung tissue, most frequently advanced tuberculosis, and usually show the alveolar arrangement. Staining is quite unnecessary, the only difficulty being the possible derivation of the fibres from retained

Stain.

Cells.

Pigment.

Crystals.

Elastic tissue.

*To test sputum for albumin Sahli presents Wagner's method, viz.:—Treat with 30% acetic acid and shake thoroughly to precipitate mucin, filter, wash, treat filtrate with NaOH until nearly neutralized and test for albumin by heat, potassium ferrocyanide or Esbach's method.

†Boiling equal parts of sputum and 10% Na O H or KOH, setting aside for 24 hours and then examining selected portions of the precipitate is a useful device and search is seldom successful without it.

food in specimens not properly safeguarded in collection. Owing to he presence of a peculiar ferment *they are often absent in pulmonary gangrene.* (f). *Leucocytes.* Eosinophiles are abundant in asthmatic sputum and neutrophiles in all sputa. They may show phagocytic inclusions of fat, carbon or hematoidin. (g). *Erythrocytes.* Microscopic blood exists in most sputa associated with cough of any marked degree, but macroscopically it is present only in violent paroxysmal cough, accidental hemorrhage and true inflammatory or necrotic processes. Fibrin if present may be demonstrated by Weigert's method. (G). *Crystals.* The rhomboidal crystals of cholesterin indicate emphysema or lung abscess, seldom phthisis. The sharp slender fasciculated fatty acid crystals suggest gangrene or advanced tuberculosis, while the colorless, octahedral Charcot Leyden crystals point to bronchial asthma. Yellow or brown amorphous masses or rhomboidal crystals of hematoidin point to blood retention and ulceration within the alveoli, and leucin and tyrosin may be present in emphysema. (h). *Bacteria.* Among those occurring in sputum are:—*tubercle bacilli* (and its simulators the smegma and timothy bacilli) and those of pneumonia, anthrax, influenza, typhoid, glanders, plague and leprosy as well as the Friedlander bacillus, micrococcus tetragenous and others. *The tubercle bacillus is by far the most important and for its determination most careful work and even animal inoculation may be necessary.* In most instances it is easy to find the germ in the cheesy particles or thicker portions of a specimen, but at other times they must be obtained by centrifugalization of the sputum.

Blood cells.

Crystals.

Bacteria.

Tubercle bacillus.

To Procure a Concentrated Sediment.—Take of sputum one part, distilled water ten parts and stir in repeatedly while boiling a few drops of sodium hydrate solution (10)%—when practically homogeneous the mixture may be directly centrifuged and examined—but it is much better to add a few drops of a phenolphthalein solution and add acetic acid drop by drop, while stirring, until only a faint pink remains. The resulting sediment may then be centrifuged and examined, some of the original sputum being mixed with the sediment to secure better fixation in the staining process. *The bacilli are often readily found in sputum thinned by spontaneous decomposition.*

Thinning.

Centrifugal method.

The stain commonly used is the Ziehl-Neelsen (saturated alcoholic solution of fuchsin or gentian violet added drop by drop to 5% solution of carbolic acid until a surface sheen appears) which keeps indefinitely as does Czapelewsky's solution (1 gm. fuchsin to 5 c.c. of liquid carbolic acid, add 50 c.c. of glycerine stirring thoroughly and finally add 100 c.c.

Stains.

of water). To eliminate the less resistant smegma or timothy bacillus the specimen should be thoroughly decolorized by Czapelewsky's method and Ebner's fluid (hydrochloric acid gms. 2.5, sodii chloride 2.5, distilled water 100, alcohol 500), or by Pappenheim's solution which not only decolorizes the smegma bacillus but stains it blue (absolute alcohol 100 c.c.+1 gm. corallin+methylene blue to saturation). The actual procedure would be (a). *Place on coverslip small portion of selected sputum.* (b). *Spread it.* (c). *Dry thoroughly in the air.* (d). *Heat* by passing it deliberately but steadily through alcohol or small bunsen flame from 6 to 8 times. (e). *Stain* with the fuchsin either by holding the stain-covered coverslip over a flame for three minutes or placing it smear side down upon the surface of a Petri dish containing stain and heating *to* boiling point (steam and rising of small bubbles) for same period. (f). *Pour off dye and add corallin decolorizing solution,* repeating and slowly draining it off five or six times. (g). *Wash thoroughly.* (h). *Mount in balsam.* The older direct decolorization method of Gabbett is coarser and involves the risk of decolorizing a certain number of the tubercle bacilli. *The greatest patience is required in these examinations and diagnosis should never be based upon a doubtful finding. Wherever there is one germ more can and must be found* and terrible mistakes have resulted from hasty conclusions though far less deadly than the too common failure to find the germ when present.

Best method.

Caution.

DISEASES OF THE NOSE, PHARYNX AND LARYNX.

This modern specialty has the advantage of permitting practically all diagnoses to be made at sight through the use of special appliances for that purpose. The larynx and post-nasal space are readily brought into view by the use of light reflected by a concave mirror upon the surface of a small plain mirror which receives the image of the structure to be investigated. The various forms of laryngoscopes are too familiar to need detailed description.

Diagnosis at sight.

Laryngoscope.

Technique.—The essentials are (a). *Strong, steady light and a good reflector.* (b). *Properly heated laryngoscopic and rhinoscopic mirrors.* (c). *A steady hand and thorough knowledge of the anatomy of the parts.*

Essentials.

The long handled mirrors are held in the pen position and in the case of the larynx are introduced without touching any structure until they press back the soft palate. The tongue is grasped in a dry napkin between the index finger and thumb of the unoccupied hand and held firmly forward. The index finger should rest against the lower teeth forming a support to steady the hand and prevent painful pressure

Technique.

upon the median raphé. If the mirror be steadily held at the upper angle an excellent view of the larynx is obtained, the upper portion of the image representing anterior, the lower, posterior structures. Deep *breathing* and *voice production* reveal the action of the vocal cords. In this examination the attachment of the handle to the mirror is above.

Vocal cords.

Naso-pharynx.

In examining the naso-pharynx a smaller mirror is used and introduced well back to the posterior pharyngeal wall, the free edge of the mirror being upward. If the patient by deep breathing and passivity can maintain a relaxed soft palate the parts may be seen without difficulty, but oftentimes some one of the special forms of the palate retractors must be used to pull that structure firmly forward and slightly upward. *Hesitancy, fumbling and tremor* on the part of the examiner invariably cause retching, and occasionally hyperæsthesia may be so marked as to necessitate the preliminary administration of a bromide or the use of a dilute solution of cocaine. *The small mirror should invariably be surgically clean, warmed to prevent misting, and tested for excessive heat by laying its back against the skin of the hands.* The patient's head should be slightly tilted backward to best show the larynx and held perpendicular or inclined forward in examining the naso-pharynx. The anterior nasal chamber is examined by direct light, the patient's head being tilted backward and the alæ drawn outwards by means of a probe or one of the various nasal dilators, bringing into view the vestibule, septum, inferior meatus, inferior turbinate, middle meatus and middle turbinate. Obstructing secretion is sprayed or wiped away and congestion is readily reduced by cocaine or suprarenalin.

Obstacles to examination.

Position of head.

DISEASES OF THE NOSE.—Acute Coryza (acute rhinitis).—A description of this universally known ailment is unnecessary. The susceptibility of an individual is markedly increased by the existence of a chronic catarrhal inflammation of the nasal passages, exhaustion, chronic diseases and lowered vitality from whatever cause. The mucous membrane in such cases shows primarily, congestion, swelling and diminished secretion, secondarily, the characteristic profuse mucopurulent discharge.

Chronic Rhinitis (chronic catarrh).—Resulting from repeated acute attacks or due to general impairment of nutrition, presents much the same appearances as acute rhinitis.

Turbinate hypertrophy.

Hypertrophic Rhinitis.—By chronic catarrhal inflammation the vascular turbinate tissues become permanently distended and the epithelial layer thickened and infiltrated. This condition is frequently associated with exostoses or ecchondroses of the nasal septum and is

most marked at the anterior and posterior portions of the middle turbinate. The symptoms are chiefly those of chronic nasal obstruction and catarrh, which lead to mouth breathing and frequent acute or subacute attacks. There is a troublesome recurrent obstruction especially marked in recumbency, such patients awaking in the morning with dry lips, coated tongue and oftentimes foul breath. The drainage of secretion is interfered with and may cause obstruction of the nasal duct; the senses of smell, taste and hearing may be markedly affected, and the Eustachian tube may be the seat of a catarrhal process. Headaches are common either as the result of infundibular occlusion or actual disease of the frontal sinus.

Chronic rhinitis.

Mouth breathing.

Deafness common.

Headache.

Exostoses and Ecchondroses.—These formations are recognized at sight and their consistence determined by the use of the probe.

Septal Deviations.—This condition, rarely absent in slight degree, is pathologic only when it produces pressure or absolute obstruction. Marked deviations lacking a history of traumatism are usually associated with a high arched palate and probably with previously existing adenoid growths of the naso-pharynx. They may be partial or general, angular or curved, are readily recognized upon examination and usually associated with chronic catarrh.

Septal Hæmatoma.—Usually the result of an injury, it is recognized as a blood red tumor.

Septal Abscess.—Is a frequent source of septal perforation and forms a fluctuating swelling.

Atrophic Rhinitis (dry catarrh).—This is usually a sequence of chronic hypertrophic rhinitis and is characterized by foul breath and a dry glazed mucous membrane carrying scanty and foul secretions which dry into adherent and offensive scabs and scales. Not infrequently, there is ulceration of underlying tissues and in long standing cases, actual structural atrophy, the turbinate bones being shrunken and the septum thin.* In delicate children the condition may coexist with a thin watery excessively fetid secretion (ozæna).

Foul breath.

Necrosing Ethmoiditis.—As a result of inflammation of the middle turbinate, necrosis of the inferior turbinate and other ethmoidal structures may occur. Periostitis and necrosis are usually followed by the exfoliation of a limited amount of bone. The symptoms are those of a suppurative inflammation specially involving the middle turbinate. Pressure deviation of the septum is common and nasal polypi frequently

*The nasal chambers look extraordinarily and characteristically roomy in such cases.

result and may remain as evidence of past disease. The probe introduced at the site of the suppuration usually detects the necrotic bone.

Hay Fever (periodic vasomotor coryza).—However obscure the causal factors, the disease offers no diagnostic difficulties. The symptoms are merely those of an unusually obstinate or persistently recurrent acute coryza with a profuse watery rather than muco-purulent discharge, coming on periodically often at a stated day or week in each year and not infrequently associated, alternating with, or substituted by, attacks of pure bronchial asthma. The disease is most frequent in the later spring months (rose cold) and in August and September (hay fever) and in some instances occurs irregularly or following exactly such influences as are described under the exciting causes of asthma. In certain cases a violent harassing and markedly paroxysmal cough may be present. Slight fever may occur at intervals and the usual duration is from four to six weeks.

Periodicity.

Nasal Polypi.—These may be either mucous or fibrous, the former being soft and gelatinous, the latter hard. They are most common in the posterior chamber, may cause necrosis, invade the adjacent cavities, deform the nose and in the case of fibrous growths may become sarcomatous.

Sarcoma and Carcinoma.—The sarcomata are usually sessile, light red in color and associated with ulceration and offensive discharge. Carcinoma is usually epitheliomatous or encephaloid in type and is excessively rare as a primary disease.

Nasal Syphilis.—Primary lesions are almost unknown, secondary lesions coincide with the appearance of the exanthemata upon other parts, but tertiary lesions appear as nodular swellings on any part of the nose. Behaving here as elsewhere, they undergo absorption or necrosis, attended by a foul breath and a rapidly destructive necrosis of all tissues. No more terrible picture is presented than that of rapidly progressive syphilitic necrosis involving the nasal structures.

Widely varying causes.

Epistaxis.—*Nasal hemorrhage occurs chiefly as a result of excessive physical and mental exertion, injuries, operations, erosions, ulcerations, tumors* affecting the nasal mucous membrane, *venous obstruction* as in tumors of the throat and mediastinum and in *mitral and tricuspid disease or high arterial pressure associated with left ventricular hypertrophy or arterio sclerosis.* Nasal congestion alone or as a part of a cerebral congestion gives rise to single or repeated hemorrhages and it occurs as a prodromal symptom of acute infectious diseases, especially typhoid, and is a complication of profound dyscrasias such as leukæmia,

hæmophilia, scurvy and purpura, in which it may be fatal. Rarely it is a source of vicarious menstruation or hemorrhoidal bleeding, and it should be remembered that repeated hemorrhages often disregarded or overlooked in case-taking may be the source of a profound secondary anæmia.

A source of anæmia.

Foreign Bodies in the Nose.—In a child or insane person a mucoid discharge becoming purulent or perhaps fetid and bloody should suggest a foreign body in the nose, and demands a rhinoscopic examination.

DISEASES OF THE PHARYNX.—*Acute, chronic and atrophic pharyngitis* present essentially the same symptoms as do similar lesions of the nasal mucosa. In the acute form the throat is sore, stiff and dry. There is dysphagia, hoarseness and in severe cases swelling of the cervical glands, the patient is continually trying to clear the throat and the secretion tends to become viscid and tenacious. Chronic pharyngitis presents much the same symptoms during its period of exacerbation and frequently shows a special involvement of the follicles, giving the throat a granular appearance, sometimes described as a separate disease, "follicular pharyngitis." The atrophic form shows a thin glistening mucous membrane, and the throat is dry and stiff and may be dotted with islands of dried secretion. The naso-pharynx may be chiefly involved leading to a dry burning sensation and most troublesome and irritating efforts to dislodge secretion (hawking). In acute conditions the secondary increase of secretion may be profuse. The atrophic form is especially disagreeable in this region and a hypertrophic form may involve the lateral walls of the pharynx.

Post-nasal Adenoids.—These lymphoid structures greatly affect the health, physical and mental development and future life of children. They represent hypertrophied lymphoid tissue and form vascular growths either broad based or sessile, sometimes moderately firm and fibrous. They are astonishingly frequent and may affect whole families. Excessively cold, or damp and changeable climates seem to increase their frequency and though the condition may be congenital it is seldom noticeable until the age of two years. The symptoms are varied and interesting. In the typical and pronounced cases the child is a mouth breather with the physiognomy shown in our illustration, p. 120, and in such cases the growth is usually large. Smaller bodies may nevertheless produce marked irritability, restlessness and a complete change in the disposition and mental activity of the child. In all cases there is increased liability to acute coryza, croup, tonsillitis, laryngitis and bronchitis and added danger from attacks of measles, scarlet fever, whooping

Importance.

Frequency.

Climate.

Symptoms.

Mouth breathers.

Mental effect.

Invite disease.

Sequelæ.

cough and diphtheria. If unrelieved they may undergo atrophy as adult life approaches, usually leaving behind chronic naso-pharyngeal catarrh, high arched palate and a deformed chest. Mouth breathing, nasal voice and snoring and the evident increase of obstruction in the dorsal recumbent position should call attention to these cases. Impaired hearing is extremely common and is due to involvement of the Eustachian tube. It is moreover one of the reflex causes of asthma,

Reflex cause of disease.

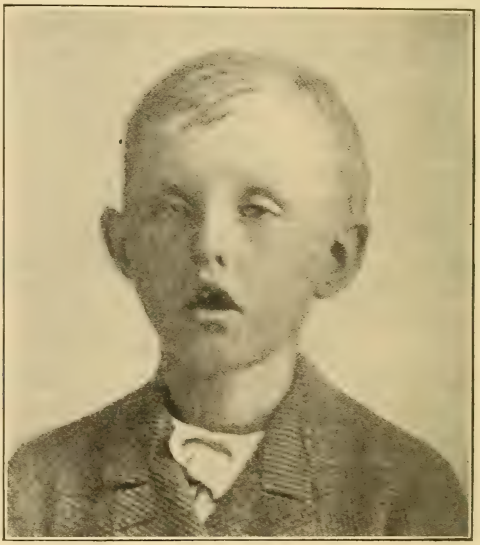


Fig. 46.—Facies of adenoid disease. (Courtesy of Dr. J. E. Schadle.)

enuresis, night terrors, teeth grinding, gastro-intestinal disturbances, croup and possibly epilepsy and chorea.*

Syphilitic Pharyngitis.—A secondary erythema is diffuse or takes the form of a *vertical red band* on the anterior pillars, which ends abruptly at the uvula. An accompanying laryngitis is usually present. Mucous patches if present tend to symmetry but tertiary lesions are frequently unilateral.

Usual site.

Retro-pharyngeal Abscess.—Such abscesses are unilateral and most often in the oro-pharynx, but may be opposite either the nasal or

* The illustration shows a marked case presenting all of the typical symptoms enumerated above, but after operation taking on a normal development and facial expression.

laryngeal portion and are often to be seen only with the mirror. Injury and caries of the vertebræ are the usual causes though they may complicate tonsillar abscess, syphilis and tuberculosis. According to their location they cause obstructed nasal breathing, spasmodic dyspnœa, deafness, or tinnitus aurium, combined with symptoms of severe pharyngitis and the local and general symptoms of abscess.

Causes.

Symptoms.

Paralysis and Tumors.—Paralysis of the pharynx need not be separately considered, the tumors of this region may take any form and belong to surgery.

DISEASES OF THE TONSILS.—Acute Tonsillitis.—This common disease of young people is rare in infants and the elderly. Its relation to acute rheumatism, chorea, endocarditis and erythema nodosum is extremely suggestive. It prevails chiefly in the spring in this country and affects both sexes equally.

Age.

Related ailments.

Symptoms.—A chill or chilliness, muscular and bone pain are followed by rapidly rising fever, with sore throat and dysphagia; the temperature reaches 103–105° F., and the voice may be thick and nasal. The tonsils are swollen and *dotted* with a readily *detachable* exudate, which may become confluent, but tends to be limited to the tonsil. The disease lasts but a few days and is usually uncomplicated. In certain instances the diphtheric membrane may be so closely simulated as to demand cultural tests.

Exudate.

Suppurative Tonsillitis (quinsy).—This differs from the preceding form chiefly in the *predominating* involvement of one tonsil, more violent onset, higher fever and pulse rate and greater prostration. The local symptoms are those of pain, dysphagia and marked swelling of both the tonsil and the peritonsillar tissues. The cervical glands are enlarged, salivary secretion is increased and within two or three days fluctuation is evident and through rupture or by the use of the knife the disease passes. Both tonsils may be involved, the pus may burrow with unexpected rapidity, and edema of the glottis may occur. *Quinsy should not be allowed to go on to spontaneous rupture.*

Severe ailment.

Dangers.

Chronic Tonsillitis.—Chronic hypertrophy often associated with dense adhesions, like naso-pharyngeal adenoids is closely associated to so-called lymphatism and the significance and effect of the two lesions is essentially the same (see adenoids).

DISEASES OF THE LARYNX.—Simple, acute and subacute laryngitis are too well known to need extended description, their chief characteristics being varying degrees of hoarseness, painful and distress-

Common and well known.

ing cough, general congestion of the mucous membrane and vocal cords and scant viscid secretion.

A complication sequel or neurosis.

Chronic laryngitis is a common complication of other diseases of the respiratory tract and a very frequent form of *fatigue neurosis* such as is experienced by singers, auctioneers, hucksters, army officers and others. In its milder forms it is associated with the excessive smoking of tobacco, overuse of alcoholics, and the rheumatic or gouty diathesis.

Etiology.

It is particularly frequent in all poorly nourished persons, especially such as live or work in dusty, steamy or vitiated atmospheres. Chronic or recurrent hoarseness of varying degree, scanty tenacious secretion, local irritation, laryngeal congestion and more or less cough are the chief symptoms.

Symptoms. Cords.

The vocal cords are sometimes unaffected and even whiter than normal, though occasionally their movement is restricted by swelling of adjacent structures.

Cough.

The cough may be paroxysmal or violent and projectile, the dried secretion acting like a foreign body.

Atrophic form.

A form precisely similar to *atrophic* rhinitis and pharyngitis may occur.

Edema of the Glottis.—Cases of sudden onset and rapidly fatal termination may occur but usually it is preceded by progressive inspiratory dyspnœa with hoarseness or aphonia.

Prompt relief demanded.

The laryngoscope or even the finger at once reveals it and no condition more urgently demands prompt, often immediate, and radical treatment. It should be remembered that this condition may complicate actual traumatism, especially burns, or the inhalation of poisonous and irritating gases, the ingestion of corrosive poisons, diphtheria, erysipelas, quinsy, scarlet fever, typhus, Bright's disease, syphilis, tuberculosis and diabetes mellitus. It is very rare in association with primary acute laryngitis.

Croup.—This well nigh universal ailment of children occurs between the ages of six months and three years, rarely later, and is undoubtedly a purely reflex condition, rickets, the irritation of teething, large tonsils and adenoid growths being the chief factors.

Reflex dyspnœa.

The symptoms are those of spasmodic inspiratory dyspnœa. It is preceded by a characteristic hoarse, brazen, hollow cough, accompanied by marked hoarseness and during the spasm the child is cyanotic and asphyxiation may seem imminent. The peculiar crowing sound attending the forced indraft of air through the glottic chink is characteristic of the disease and familiar to both physician and layman. In many cases the attacks last only for a short time, usually coming on about midnight, in others they may recur on several successive nights and the child is hoarse and has a croupy cough.

Laryngismus stridulus.

Convulsions rarely occur and death during the attacks is almost unknown. *Laryngismus stridulus* is a terma pplied to an

adductor laryngeal spasm which may be either nocturnal or diurnal and lacks the hoarseness and cough of croup. It corresponds to "passion fits" "child crowing," "holding the breath" of the laity and convulsions, carpo-pedal or general, may occur.

True Croup (membranous croup).—In rare instances especially in measles and pertussis this terrible disease is unassociated with diphtheria, but ordinarily any persistent laryngeal stenosis suggests a diphtheritic membrane and demands prompt intubation and large doses of diphtheria antitoxin.

Usually diphtheria.

Syphilitic Laryngitis.—This is ordinarily a tertiary lesion and may occur two or three decades after the primary infection. The secondary stage may however give rise to hyperæmia, symmetrical ulceration and condylomata which may resolve or ulcerate. The gumma may cause deep ulceration or necrosis with resulting stenosis from cicatricial contraction. The therapeutic test is often necessary. Congenital laryngeal syphilis usually appears in infancy, more rarely at the age of puberty (hereditaria tarda).

Usually tertiary.

Therapeutic test.

Tuberculous Laryngitis.—Primary tuberculosis in this region is extremely rare and usually though not always the lesion indicates an *advanced* pulmonary lesion.* The symptoms are those of a persistent chronic laryngitis, hoarseness or aphonia being a marked and early symptom and swallowing painful if there is epiglottic or pharyngeal ulceration. The laryngeal mucous membrane is at first *pale* and later an *ashy gray, the arytenoids show a pyriform swelling and the epiglottis is turban shaped. The ulcers themselves are shallow and broad with gray bases and irregular outlines, and the vocal cords usually appear "moth eaten" from ulceration.* The tubercle bacillus can usually be demonstrated easily and makes the diagnosis positive.

Seldom primary.

Hoarseness, cough and aphonia.

Ashy gray color.

Pyriform swelling and epiglottic turban.

Moth eaten cords.

Tumors of the Larynx.—The general symptoms of laryngeal tumors are dysphonia and aphonia, intermittent or persistent dyspnœa, cough, and sometimes laryngeal spasm. Certain growths above the cords are subjectively symptomless and some sessile growths produce strikingly intermittent and irregular symptoms with violent paroxysmal cough and are often difficult to see with the laryngoscope.

DISEASES OF THE BRONCHI, LUNGS AND PLEURA.

ACUTE AND CHRONIC BRONCHITIS.—Etiology.—The etiologic factors are essentially the same in all forms of simple bronchitis.

*Nearly all of the supposedly primary cases prove to be secondary to advanced but latent or partially arrested pulmonary lesions. They will become still rarer as the X-Ray is more generally used in diagnosis.

Climate. A changeable, damp climate, whether temperate or tropic, is a potent factor and general low vitality invites it. Chronic gout, alcoholism, Bright's disease, incompensated mitral lesions and malaria predispose to it and it invariably accompanies tuberculosis, emphysema and asthma. In acute disease such as measles, typhoid, smallpox, influenza and whooping cough it is a common or invariable complication. Males are chiefly affected by reason of their greater exposure to climatic influences and the diseases of etiologic relationship, and it is particularly prevalent at the extremes of life. Steel grinders, stone cutters, bakers, millers, stokers, felt workers, and employees in flax and cotton mills show a special tendency to this disease but so far as acute bronchitis is concerned the common "coryza" is ordinarily the exciting factor, the disease extending apparently by continuity of structure from the upper to the lower air passages. The mistaken and foolhardy process of "hardening" young infants by reckless exposure to harsh weather is a potent factor in the production of the disease in infants and young children, and helps greatly to swell infant mortality.

Previous health.

Antecedent diseases.

Sex.

Age.

Occupation.

Coryza.

"Hardening" infants.

Morbid Anatomy.—At first, in acute bronchitis, the mucous membranes are inflamed, dry and irritable, later there is muco-purulent secretion, the membranes are swollen, may be edematous, and should the bronchioles become affected a disseminated broncho-pneumonia (capillary bronchitis) results. In chronic bronchitis the long continued inflammatory changes lead to permanent thickening or atrophy, granulation or even ulceration and it is one of the causes of a fusiform or saccular dilatation known as bronchiectasis.

Fever.

Cough.

Sputum.

The symptoms of acute bronchitis are *fever*, usually moderate, (99.5°–103° F.) *substernal pain, soreness or oppression, hoarseness, general malaise, cough,* at first dry, ringing, harsh and somewhat painful, later moist and hollow. The sputum, at first mucoid and scant soon becomes muco-purulent and sometimes profuse, and if the cough is violent may show blood streaks.

Râles chiefly.

Physical Signs.—The only important physical signs are the *sibilant and sonorous râles* of the first stage followed by the *mucous, bubbling or even gurgling râles,* of the stage of exudation. Slight dyspnœa and cyanosis *may* be evident, and palpation may reveal rhonchal fremitus. Marked cyanosis suggests disseminated broncho-pneumonia, a preexistent emphysema, or a complicating cardio-vascular lesion.

Brief duration.

Prognosis.—It lasts from a few days to several weeks and one may ordinarily anticipate a prompt and complete recovery and freedom from

relapse save in those persons exhausted by antecedent disease or badly nourished.

Symptoms of Chronic Bronchitis.—This is essentially a *secondary* disease; the hereditary element may be marked; it is an invariable accompaniment of asthma, incompensated mitral disease or emphysema and many persistent cases are associated with chronic diseases of the kidneys. The diagnoses of "chronic bronchitis" and "winter cough" have of late years diminished, as exact methods have proven many, especially in the young, to be cases of pulmonary tuberculosis. *Putrid bronchitis* is chronic and characterized by foul smelling sputum, but hardly deserves a separate place because of its almost invariable association with bronchiectasis, gangrene, and a ruptured abscess, or empyema.

Fundamental points.

Putrid bronchitis.

Physical Signs.—Unless, as is commonly the case, one finds well marked symptoms of emphysema, the physical signs of this ailment are limited to sibilant, sonorous, mucous or bubbling râles with more or less profuse expectoration.

Chiefly auscultatory.

In fibrinous bronchitis fibrinous casts are formed and expelled; these are often extensive and sometimes produce bronchial blocking and atelectasis of lung areas. Dyspnœa and cough are more urgent than in ordinary bronchitis and the attacks may be single, recurrent, acute or chronic. It is a *clinical curiosity*.

Fibrinous casts.

Urgent symptoms.

DISEASES ASSOCIATED WITH CHRONIC BRONCHITIS.

EMPHYSEMA.—This may be acute or chronic, general or local, interstitial or vesicular, and a vesicular emphysema may be either atrophic or hypertrophic. A localized emphysema may be temporary or permanent and is usually vicarious, representing the effect of over-action associated with temporary or permanent loss of function in other portions of the lungs. The best examples are seen in pulmonary tuberculosis. *Interstitial Emphysema*. This condition is caused by the rupture of a superficial or deep air vesicle which, during violent cough or straining, permits the escape of the air into the areolar tissues and produces either (a) *interlobular or* (b) *subcutaneous emphysema*, or even (c) *pneumothorax* according to the site and degree of its extension. *Subcutaneous emphysema* is especially common in connection with traumatism involving the lung structure and is at once detected by the exquisite crackling produced by the pressure of the fingers upon the distended skin. *The atrophic or small lunged emphysema* hardly merits a special description and is essentially a senile condition.

Varieties.

Vicarious.

Ruptured intervesicular septa.

"Crackling."

The *common form of emphysema* is the vesicular hypertrophic. In this variety the air cells are overdistended, their walls weakened and prone to rupture and, as a result, the lung is increased in size, its alveoli tend to become fused in certain areas and its circulation and expansile power are greatly affected.

Bronchitis, dyspnœa and cyanosis.

Symptoms.—The symptoms are primarily those of a chronic bronchitis plus slight or intense cyanosis, dyspnœa, persistent or developed by exertion, and certain well defined and easily recognized physical signs. A considerable degree of emphysema may exist without dyspnœa or persistent signs of bronchitis, yet it represents the most profound cases of cyanosis seen in adult walking patients. *Cough.* Cough may be variable in degree as in persistence and may be dry, moderately productive, or accompanied by profuse expectoration. In severe cases it is somewhat characteristic, each individual paroxysm being greatly prolonged *diminuendo*.

Fig. 47.—Emphysema. Attitude frequently assumed, concealing barrel form of chest.

Barrel chest.

Chest movement.

Inspection.—The shape of the chest represents that of forced held inspiration (barrel chest). The anteroposterior diameter is increased, the epigastric angle wide, true expansion is largely replaced by a vertical lifting movement, and constant overuse makes prominent the accessory

Fig. 48.—Emphysema. Same chest, patient in dorsal recumbent posture.

muscles of respiration, particularly the sternomastoids. The ribs are rigid, the interspaces broadened and the expiratory descent of the thorax is prolonged. The apex beat of the heart is ordinarily invisible, the neck appears short and a compensatory curve of the spinal column may cause the chest to appear flattened and the patient round-shouldered. If such a one be placed upon the back on a table, the chest will at once

Apex beat obscured.

assume its typical emphysematous outline. Epigastric pulsation is common and Litten's sign shows an abnormally slight range of diaphragmatic excursion confirmed by the fluoroscope which also shows abnormally bright lungs. Useful manœuvre. Litten's sign.

Palpation.—Palpation confirms inspection and reveals enfeebled fremitus and apex beat and usually a liver displaced downward.

Percussion.—Percussion develops hyperresonance, low position of the lung border, impaired movement of the lung bases and apices, displacement of the liver and diminution or entire absence of the superficial cardiac area.* Low borders.

Auscultation.—The breath sounds are enfeebled and expiration is prolonged though usually *low* pitched. Bronchial râles are ordinarily present at the base, the heart sounds are muffled, especially in the mitral area, but the pulmonary second sound though enfeebled may show relative accentuation. In old and severe cases tricuspid regurgitation may be present. Enfeebled sounds.

Secondary Symptoms.—The effect of long standing severe emphysema is to over-burden the pulmonary circulation and right heart, hence if incompensation follows one may expect to find the symptoms of a cardiac lesion. As a matter of fact such severe terminal symptoms are rarely encountered though minor degrees of secondary congestion affecting the organs tributary to the great veins are extremely frequent. Cardiac strain.

FOREIGN BODIES IN THE BRONCHI.—Coins, whistles, nails, pins, tacks, buttons, jewelry, bullets, pebbles, false teeth, tooth crowns, bones, fruit stones, peas, beans and the like may be drawn into the glottis and usually fall promptly in the larger right bronchus. Violent cough and air hunger or true stenotic dyspnœa and cyanosis usually follow and are associated with widely varying degrees of pain. If the object is not expelled death may result in a few minutes but more often the attack subsides and secondary inflammation, often suppurative, ensues and results in localized septic broncho-pneumonia, abscess, or even gangrene. In some instances chronic pneumonia with or without bronchiectasis results and lasts for months or years ending rarely in the extrusion of the foreign body. Immediate symptoms. Sequelæ.

Diagnosis.—This depends upon the anamnesis, violent paroxysmal cough, the Rœntgen ray findings and the physical signs of localized inflammation often associated with airless areas representing atelectasis. Usually simple.

BRONCHIECTASIS.—Rarely congenital, it ordinarily follows

*In certain cases marked dulness may be present at an apex and in others the general percussion note is not clearly hyperresonant.

chronic inflammation of the bronchial wall alone or with chronic obstruction and accumulation of secretion, as in pulmonary tuberculosis, chronic pleurisy with adhesions and retraction or fibroid disease of the lung. Though rarely associated with acute diseases, it has been reported in influenza and broncho-pneumonia.

Physical Signs.—*Often absent, they, when present, are merely those of lung cavity, usually lacking the distinguishing qualities of a vomicus occupying infiltrated tissue.* Hence *the diagnosis of bronchiectasis usually rests upon the presence of causative factors, the peculiar paroxysm of cough and the characteristic sputum.*

Cough.—Once or twice daily, usually in the morning on arising, a violent paroxysm of coughing occurs attended by extraordinarily profuse expectoration. This is offensive in odor, gray or grayish brown and purulent and upon being placed in a glass it will after a time separate into three layers. The lowest is thick and granular; there is a thin mucoid intermediate layer, and the surface layer is brown and frothy (high air content). Changes of attitude may greatly influence the onset of cough, and to empty the bronchiectatic cavity some patients assume an attitude that brings the head below the body.

Typical sputum.

Prognosis.—The condition once established is permanent, progressive, and rarely relieved save by operative measures.

SPASMODIC ASTHMA.—Definition.—*A respiratory neurosis of ill-defined causation characterized by more or less severe paroxysms of expiratory dyspnœa, tending to chronicity and associated with bronchitis and emphysema.*

Etiology.—It is often closely associated with, or a manifestation of, hay fever, but many cases show bronchitis, polypi, adenoids and supersensitive or hypertrophied turbinate bodies, intestinal auto-intoxication or irritation, malnutrition, hysteria, neurasthenia or epilepsy.

Exciting Causes.—Careful inquiry should be made concerning the exciting causes and the number and variety of the attacks. Such patients become close observers and can often describe with exactness the clinical phenomena and apparent exciting cause. Amongst these are *odors* of all sorts; those of drugs, of certain flowers, of hay, of horses or dogs or cats, fish, game or coffee; *irritants* such as dust or fog; *heat or cold*, whether local or general. Old feather beds and pillows have in more than one instance proved causative factors and *emotions* such as pleasure, pain, joy and sorrow, sexual stimuli, grief and worry are alike potent. In certain instances *heavy meals at night* and certain articles of food or drink may cause a seizure and in some cases it is

Curious factors.

distinctly related to the menstrual epochs. Chronic poisoning, such as that from gout or lead, intestinal worms, and various occupations exposing their followers to dust or alternations of heat or cold may excite attacks.

A Word of Warning.—The utmost care should be observed to avoid the common and disastrous error of confounding the so-called "renal," "aneurismal" and "cardiac" asthmas with those of the ordinary spasmodic type. *The paroxysms of "renal asthma" are absolutely like those of true asthma* and the cause can only be determined by a careful examination and quantitative analysis of the 24 hours urine, for the attacks are usually coincident with low urea excretion. The pseudo-asthmatic paroxysms produced by aneurism of the aortic arch may deceive the elect, but are fortunately rare. *Cardiac Asthma.* Ordinary cardiac dyspnœa is either persistent or distinctly dependent upon exertion and is inspiratory in type or a mere air hunger. Nevertheless true asthmatic attacks occur as the result of weakened hearts and in established asthma the heart strength is an important factor in the treatment and prognosis.* Males are chiefly affected, the ratio being as two to one, and 80% of all cases appear in persons under 40 and ⅓ in those under 10. *It is distinctly an hereditary alternative.*

Renal and cardiac asthmas.

Exertion dyspnœa.

Theories as to Causation.—Five may be mentioned. 1st. *Bronchial spasm*, i.e. spasmodic contraction of the muscles controlling bronchial caliber. 2nd. *Hyperæmia and swelling of the mucosa.* 3rd. *Spasm of the diaphragm.* 4th. *Spasmodic contraction of other inspiratory muscles.* 5th. *Exudative bronchiolitis.* Bronchial spasm (1), congestion and the attending exudation (2 and 5) are undoubtedly the most prominent factors, while it is not impossible that all five may be operative in varying degrees.

No *one* causative.

Time of Attack.—Like croup, angina and nocturnal epilepsy, attacks of asthma occur usually at midnight or in the early morning hours.

Auræ.—Like epilepsy attacks of asthma may be preceded by premonitory symptoms recognized by the patient. Amongst these are mental depression or exaltation, flatulence, yawning, sneezing, itching, headache and polyuria. Ordinarily, however, the attacks occur without any such warning.

*In a case recently observed typical asthma of long standing was promptly and completely relieved by treatment directed wholly to a dilated heart and this experience is a common one.

Onset. **Symptoms.**—The onset may be gradual, but is usually sudden. There is a sense of constriction of the throat or chest and pronounced dyspnœa with difficult, stridulous and prolonged expiration. In spite of the frenzied gasping the *rate* is usually below normal. A short, dry, feeble cough increases the patient's discomfort, and an acute emphysema enlarges the girth of the lower chest so that the female patient, if attacked when dressed, is obliged to loosen the clothing. The eyes are bloodshot and staring, the voice gasping and weak, the face pallid, cyanotic, drawn and agonized. The mind is absolutely clear and the patient appreciates his suffering and is usually apprehensive and fearful. If in bed, the knees are drawn up, the shoulders advanced, the back rounded, and the head is on the hands, or, sometimes one will be found leaning over a chair or bed-post or thrown back with raised arms grasping some support that will give added purchase to the accessory muscles of inspiration. In many cases patients rush to an open window, regardless of extremes of temperature.

Dyspnœa.

Facies.

Attitude.

Examination of the Chest.—*Inspection* shows an increased basal girth, dyspnœa, defective movement, inactive diaphragm, costal breathing and rigid abdominal muscles with which the patient is seeking to reinforce the respiratory muscles. *Palpation.* The râles are easily felt and vocal resonance and fremitus are enfeebled. *Percussion and Auscultation.* The signs are those described under emphysema with bronchitis stridor superadded.

Cough.—This is ordinarily feeble and unproductive until the end of an attack, when there may or may not be a considerable amount of sputum. In cases of pure asthma uncomplicated by bronchitis one may find the pearly globules of the size of a hemp seed which contain Curschman's spirals and Charcot Leyden crystals (see page 112). *Pulse.* The pulse may be markedly weak and accelerated. During any severe attack the right chambers of the heart are dilated yet death during an attack is most unusual. *Duration of the Paroxysm.* Attacks usually end gradually, less often abruptly after two or three hours, but may be shorter or much longer, usually recur with increasing severity on successive days and are followed by a period of immunity.

Spirals and crystals.

Heart.

Recurrent.

Prognosis.—The attacks are recurrent and chronic and asthmatics may endure them for a reasonably long life time, but on the average there is, nevertheless, marked shortening of life expectancy as is proven by life insurance statistics. Asthma occurring in childhood usually depends upon a removable cause and is often curable. Those cases occur-

Chronic.

Shortens life.

ring between the ages of 10 and 20 are sometimes cured, those of the intermediate period are rarely cured, and those in patients above 60 years of age, it has been said, are never cured. The author's experience would indicate that at no age is the last statement absolutely true. Careful and thorough study of causative factors may bring relief in some apparently hopeless cases but the cure of secondary conditions of long standing is never possible in the adult.

Age important.

PLEURISY (*pleuritis*).—**Definition.**—*An inflammation of the pleura, acute or chronic, with or without an exudate which may be serous, bloody or purulent.*

Applied Anatomy.—Each pleura completely invests its lung, dips between the lobes and is reflected upon the chest wall so as to form a potential closed cavity, though normally parietal and visceral layers are in close apposition and move freely over one another, lubricated by the normal lymph. The pleural cavity resembles a great lymph space structurally continuous with the diaphragm, pericardium, cervical fascia, chest wall and lung. The pleural folds or sinuses and Traube's space are discussed on page 82. *When inflamed, the pleural surface becomes dry, and the attrition gives rise to pain, cough, and the characteristic physical sign of dry pleurisy, viz.:—audible, often palpable, friction sound.* A slight attack may (a) terminate promptly without permanent damage to the pleura, (b) the fibrinous exudate may become organized and form pleural adhesions of variable extent and density, or (c) a serous exudate may result, relieving the pain by separating the pleural layers and in its turn be reabsorbed or become converted into pus (empyema). The character of the exudate varies greatly with the cause of the pleurisy and the nature of the germs present. *Bloody fluid* most often indicates malignant growths of the lung and pleura. *Purulent exudate* is usually the result of neglected effusion, but is often primary, particularly in the cases associated with the lobar pneumonia of children. If one bears in mind the important structures contained in the mediastinum, one can readily appreciate the importance of the pressure symptoms and the degree of displacement of the heart as a result of large unilateral effusions. The lung of the affected side is compressed from below upward and towards its root *pari passu* with the increase of exudate and may remain permanently crippled and bound down by adhesions after resorption of the exudate. Pleurisy is almost invariably present in lobar pneumonia, but in adults it is seldom associated with a marked degree of effusion. It is generally believed that the greater number of so-called simple pleurisies represent a local

Anatomy explains much.

Terminations.

Varieties.

Exudates.

Pressure signs.

Tuberculosis.

ized tuberculosis and the statistics of foreign observers show a percentage of tuberculous pleurisies ranging from 25–90%.*

Germs.

Etiology.—The pneumococcus, staphylococcus, streptococcus and tubercle bacillus may be found alone or in combination in cases of purulent pleurisy and to these may be added the influenza, typhoid and colon bacillus. Pleurisy may appear as a complication of various diseases. First in importance are tuberculosis, lobar pneumonia and pericarditis, but peritonitis, influenza, acute rheumatism and chronic nephritis are frequent. It may occur in the exanthemata, in typhoid fever and the acute infectious diseases generally, as well as in gout, chronic diseases of the liver and gall-bladder, and in cancer. Traumatism of the chest with or without perforation may cause it.

Associated diseases.

Symptoms of Acute Fibrinous Pleurisy.—(1). *Fever*, usually moderate in degree and lasting but a few days. (2). *Pain*, most commonly felt in the axillary or inframammary region yet sometimes not localized at the seat of the process, but referred to the terminations of the intercostals or even to the sound side. It may be fixed, stabbing or shooting in character, and is increased by deep breathing and cough. The fact that abdominal pain may be pleuritic in origin should never be forgotten for many important surgical conditions may be simulated. (3). *Cough;* this is harsh, unproductive, painful and suppressed. (4). *Friction;* auscultation yields a shuffling creaking, squeaking, or even a crackling sound simulating at times true crepitation or even rhonchi, but persisting after cough, superficial in character and usually occurring early in the inspiratory phase of a respiration. Friction is often palpable as well as audible.

Referred pain.

Characteristics.

Physical Signs.—*Inspection* reveals shallow catchy breathing, comparative immobility of the affected side, and painful cough. The patient's decubitus is variable, oftentimes he lies upon the affected side as if to limit its movement during the stage of congestion and give greater latitude to the expansion of the sound side after effusion.

Decubitus.

Diaphragmatic Pleurisy.—The pleurisy may not extend beyond the diaphragmatic surface, but usually other portions are involved. The physical signs may be absent or be those of acute pleurisy, with fever, rigid abdominal muscles, costal breathing and an extraordinary degree of pain on breathing, swallowing, talking and laughing and tenderness over the region of the 10th rib. It may be acute or chronic, dry or wet, serous or purulent and the excessive epigastric or inferior

Excessive pain.

* These are based upon the injection of large amounts of exudate into susceptible animals.

marginal thoracic pain attending diaphragmatic movement even of slight degree is the chief symptom.

Pleurisy with Effusion.—*The first effect of effusion is to separate the inflamed surfaces, relieve pain and diminish the area of friction sounds.* The fluid gravitates to the most dependent portions, and if not encapsulated by adhesions is movable and slightly or markedly changes its level according to the position of the patient and the amount of effusion. Owing to the conformation of the chest and the peculiarities of internal pressure the upper line of pleural effusion often makes an S-like curve, reaching its maximum height in the mid-axillary line. If the fluid reaches the middle of the second rib this curve disappears.

Relief afforded.

Borders and mobility.

Fig. 49.—Pneumothorax (Left) and Encapsulated Fluid (Right). Compression of the lung on left side as in pleural effusion.

Physical Signs.—Inspection.—One notes immobility of the affected side, flush interspaces,* increased dyspnœa, with cyanosis or even in extreme cases lividity, and, a displacement of the heart to the opposite side which may carry the visible cardiac impulse to the axilla on the left side or to the right mid-clavicular line. Occasionally in moderate left-sided effusion no cardiac impulse is visible.

Displaced heart.

Palpation.—Palpation confirms inspection. *Palpatory percussion* reveals *marked resistance* over the exudate. *Percussion* reveals a *flat note,* decidedly differing from that of consolidation and simulated only by pleural growths and dense pleural adhesions. *Above the level of the fluid anteriorly, the note is often hyperresonant or tympanitic* in quality (Skodaic resonance). Little difficulty is ordinarily encountered in delimiting the upper border of the effusion or in following its slow alterations of level with changes of posture. *Posteriorly, dulness due to a compressed lung may be encountered* and well above the fluid level in established effusions the unaffected tissue may yield the hyperresonance of vicarious emphysema. *Movable dulness in Traube's semilunar space* often reveals very small exudatess if light percussion is used. *The note is not typically flat but passes sharply from slight dulness to pure stomach tympany* as the patient changes from the erect to the recumbent posture.

Resistance and flatness.

Hyperresonance.

Light percussion.

Auscultation.—Over the compressed lung posteriorly the breathing

* The "bulging interspaces" are a clinical curiosity in simple acute exudative pleurisy.

is tubular but below the level of the fluid the voice and breath sounds are markedly obscured, *though seldom entirely lost.* Over the elastic chests of children and even in adults conducted breath sounds may suggest true consolidation,* though usually *of a distant* quality. Pleuritic friction may be heard above the level of the fluid, particularly if this is receding and may persist for long periods either as friction or crepitation.

Mensuration.—In large effusions the measurement of the affected side is increased.

Fluoroscopy.—The diaphragm of the affected side is lowered and shows a greatly lessened respiratory excursion and the fluid is clearly indicated by the corresponding shadow.

Rhythmic Lateral Displacement of the Heart.—In 1902, the author reported† observations based upon a number of cases of pleuritic effusion which indicated that this condition might be accurately diagnosticated by fluoroscopic methods, or even by auscultatory percussion and occasionally by inspection. He found that in the presence of a considerable unilateral effusion, the heart, in addition to its displacement, showed a distinct lateral movement of respiratory rhythm varying with the depth of respiration and the amount of effusion. In some cases, the superficial *heart impulse* could be seen to move from side to side; in nearly all, auscultatory percussion verified the observation, and in every case the fluoroscopic findings were positive. The phenomenon seems to depend chiefly upon the piston-like action of the diaphragm initiated by the thrust of the abdominal muscles. In full inspiration pressure is relieved by the descent of the diaphragm and the expansion of the chest, and the readily movable heart tends to approach the median line. In forced expiration, the rising fluid column‡ and the narrowing chest force the organ away from the median line, sometimes to the extent of two inches. The importance of the observation depends upon the fact that it it does not occur under conditions such as subdiaphragmatic abscess or in other conditions likely to be confounded with pleurisy, either serous or purulent. *The maximum range of movement occurs in medium sized effusions; in very small effusions*

* Recently the author removed over 1500 c.c. of serous fluid from an elderly patient in whom pure tubular breathing was marked throughout the area of flatness.

† New York Medical Journal, 1902. Transactions of the Association of American Physicians, 1905.

‡ In every case so far examined the upper border of the effusion was seen to rise in expiration.

there is practically no movement; in massive effusion both the chest and diaphragm are almost passive, yet in no such case so far observed has the author found an entire absence of upward thrust and lateral cardiac displacement. Hence he has not been able to confirm the statement made by certain writers to the effect that in massive pleurisies the diaphragm is *completely* immobile and may even bulge into the abdominal cavity. In every case so far observed the diaphragm on the affected side has shown movement of some degree and *must* if the abdominal muscles act strongly. *Forced respiration* is necessary and might demand the exhibition of morphia if pain were severe. In no instance so far observed has this been necessary.

Symptom constant.

EMPYEMA.—Symptoms.—To the symptoms of serous effusion are added:—*Intermittent* (septic) *temperature, sometimes a localized edema*, and, *in rare instances, actual bulging of the interspaces or the escape of pus from the pleural cavity through the intercostal space where it forms a tumor beneath the skin* and may cause an intractable fistula through spontaneous rupture. Such perforations usually occur in the antero-lateral portion of the chest. The urine may show *albumose* (see p. 325); marked leucocytosis is the rule, the average count being about 18,000 and few falling below 12,000,* and the blood may show iodophilia (see p. 394).

Wet pleurisy plus sepsis.

Blood findings.

The Use of the Hypodermic Needle.—Aspiration may sometimes be performed with an ordinary hypodermic syringe, but one usually needs one of the larger syringes of the same type with a long needle of greater calibre. Such an examination, performed under proper antiseptic precautions, is absolutely without danger even though the needle enters the lung. The physician's fingers laid upon the ribs should mark the limits of the interspace and the needle should be thrust quickly and deeply between them without touching the rib. If fluid be present and the point entered be rendered anesthetic by the ethyl chloride spray the procedure is painless. The site of the puncture is ordinarily the 5th interspace in the axillary line, but it is often necessary to enter the chest at other points representing maximal dulness, and if care be taken to avoid the heart, spleen, liver and diaphragm the operator has a wide latitude.

Danger almost nil.

Painless usually.

Pleuritic Adhesions.—A clinical diagnosis of slight adhesions is often impossible, but in general, the symptoms are as follows:—

*In simple serous pleurisy a few cases may show moderate leucocytosis during the febrile period, but in 52 cases examined by R. C. Cabot, the average count was 8,820, and in only 9 did it exceed 12,000.

Lagging and retraction.

Inspection shows impaired movement of the affected side or retraction if extensive adhesions are present and Litten's sign may show marked impairment of the diaphragmatic excursion on the affected side.

Palpation shows diminished vocal fremitus and lessened expansion.

Percussion shows a change of note varying from slight dulness to absolute flatness according to the extent and density of the adhesion.

Auscultation shows diminished breath sounds and voice conduction, and oftentimes in certain areas pleuritic râles.

Fluoroscopy.—A distinct shadow indicates extensive adhesions and the diaphragmatic movement is markedly impaired.

An obscure disease.

Interlobar Pleurisy.—This ordinarily takes the form and presents the symptoms of an interlobar abscess situated between the upper and middle lobes near the root of the lung. It presents great difficulties to the diagnostician and is ordinarily mistaken for true pulmonary abscess on account of its peculiar location. The purulent accumulation may vary from an ounce or two to a pint or more. It ordinarily follows a general pleurisy.

Usually tuberculous.

Chronic Pleurisies.—These may be dry or wet, serous or purulent, and for the most part present the same physical signs as are found in acute disease. Chronic pleurisies are usually tuberculous and the same may be said of chronic empyema, but they often represent neglected non-specific cases. Malignant disease of the pleura is attended by hemorrhagic effusion and chronic inflammation.

Course and Termination of Pleurisy.—*Simple plastic pleurisy* runs its course in a few days, leaving behind, ordinarily, a few adhesions often discoverable only at post mortem. *In cases with serous effusion* aspiration is usually required, but spontaneous reabsorption is not uncommon. Empyema ordinarily demands surgical interference and leaves behind a variable area of thickened adherent pleura, and a more or less crippled lung.

PNEUMOTHORAX.—Definition.—*An effusion of air into the pleural cavity.* An exudate is almost invariably present, and *the true descriptive terms are hydropneumothorax and pyopneumothorax*, the latter being the common form.

Usually tuberculous.

Etiology.—By far the greater number (90%) are associated with advanced tuberculosis of the lung and the formation of a fistulous communication, though in neglected empyema a fistulous opening may originate in the pleura. Amongst other causes may be mentioned penetrating wounds of the chest wall, traumatic rupture of the lung, rupture of emphysematous vesicles, pulmonary abscess, gangrene, in-

farct, cancer and even hydatids. Nearly all of the cases occur in males and the disease is invariably unilateral; sudden and complete bilateral pneumothorax should cause almost instant death.

Morbid Anatomy.—The air in the pleural cavity is ordinarily pumped in through a single valve-like opening by the respiratory movements, thus creating marked intrapleural pressure. The lung is compressed in the direction of its root, mediastinal tissues are displaced towards the unaffected side, diaphragmatic movement is almost wholly abolished, the pleura becomes inflamed and in most cases a *small amount* of purulent, rarely a serous effusion, is present.

Lung compression.

Symptoms.—*The onset is almost invariably sudden* whatever may be the cause and follows directly upon injury, physical strain, vomiting, or, more frequently, a paroxysm of cough. *Symptoms of shock are present, there is urgent dyspnœa, cyanosis and lividity, and the patient ordinarily feels that there has been some "internal rupture."* An insidious onset is rare and the author has observed but one such case. In such the opening is tiny and obstructed so that only the smallest quantities of air *intermittently* enter the pleural cavity.

Symptoms urgent.

Physical Signs.—*Inspection* shows immobility and bulging of the affected side evidence of a displaced heart and unilaterally impaired diaphragmatic movement, the physiognomy of shock, cyanosis, and evidence of marked dyspnœa. *Palpation* confirms inspection and vocal fremitus is ordinarily greatly diminished or entirely lost. The *percussion* note is ordinarily distinctly tympanitic though under conditions of extreme pressure quite a marked degree of dulness may be found. *Auscultation.* Breath and voice sounds are feeble and distant or entirely absent. If the patient be raised to a sitting posture and *gently* shaken *Hippocratic succussion* is obtained. This is merely a splashing sound readily detected if the ear is applied to the chest during the manœuvre and often heard at some little distance. An exquisite *metallic tinkling* is present in many cases as is the *coin sound*, obtained by placing the ear upon the chest wall anteriorly and having an assistant place a good sized silver or gold coin on the back and tap it with another, the conducted sound having a musical, bell-like or clear metallic ring.

Striking symptoms.

Dulness possible

Cardinal Signs.—The three cardinal signs are: *metallic tinkling, Hippocratic succussion,* and the *coin sound,* but all of the signs are ordinarily so clear that an error in diagnosis can seldom occur. *In cases where a large opening exists the signs are those of an enormous cavity with firm smooth walls* (see p. 103). In *diaphragmatic hernia*

Diaphragmatic hernia.

the history, borborygmi (rumbling, cooing) and the change of note on taking large quantities of water into the stomach or inflating it with air assist in differentiation.

Course and Termination.—The course of pneumothorax depends chiefly upon its cause, and as those associated with chronic tuberculosis (90%) are almost invariably fatal, the mortality is enormous.

HYDROTHORAX.—This is a serous pleuritic effusion without pleuritis and is a secondary ailment, usually bilateral, associated with cardiac and renal disease or terminal anæmias and cachexias.

LOBAR PNEUMONIA.—(Pneumonitis, Croupous Pneumonia, Lung Fever).—Definition.—*An acute infection characterized by primary fibrinous inflammation of the lobar type, profound toxæmia and an abrupt onset and termination.*

Germs.—Though found in normal buccal secretion and diseases other than pneumonia, Frankel's diplococcus pneumoniæ or micrococcus lanceolatus is believed to be the most frequent cause of the disease. It is present constantly in the saliva of some persons, and is found in the secondary processes of the disease. It is elliptical, the

Suggestive likeness.

lanceolates occurring in pairs and in sputum is ordinarily encapsulated.*

Its marked resemblance to the meningococcus is interesting because of the coincidence of increased pneumonia incidence and mortality with epidemics of cerebro-spinal fever.

Three stages.

Morbid Anatomy.—Three stages of the disease are recognized for purposes of convenience, but are often blended at autopsy. (1). *Congestion.* The lung is enlarged, dark red, less elastic than normal, on section frothy red liquid exudes, crepitation is impaired but sections

Congestion.

will float on water. The microscope shows marked capillary turgescence and erythrocytes, leucocytes and desquamated epithelium. (2).

Hepatization.

Red hepatization (consolidation). The appearance is as in first stage but the lung does not crepitate, is friable, and sections sink in water. The cut surface is finely granular, the exuded serum is bloody (not frothy) and minute fibrinous plugs may be scraped from the surface. The *microscope* shows a coagulated fibrinous exudate and fibrinous threads holding in mesh great numbers of erythrocytes, leucocytes, and desquamated alveolar cells. There is marked round cell infiltra-

Resolution.

tion of the interalveolar septa. (3). *Gray hepatization* (resolution).

* It is readily stained as follows:—(a) Make thin smear, dry in air and fix by heat. (b) Cover surface with the Hiss stain (gentian violet, saturated alcoholic solution 5 c.c. plus 95 c.c. distilled water). (c) Heat till steam rises. (d) Wash with copper sulphate solution (20%). (e) Dry and mount. The germ is Gram positive.

The gray color displaces the dark red of congestion and consolidation, the lung rapidly softens, becomes more crepitant, but is still friable and a purulent exudate follows the knife. As a result of the subsidence of inflammation and hyperæmia, liquefaction of the corpuscles and fibrin takes place through a digestive ferment possessed by the leucocytes, the cellular elements undergo fatty and granular degeneration, and, within from two to four weeks, absorption taking place through the lymphatics together with free expectoration removes the inflammatory products. The *duration of the stages of pneumonia* is extremely variable. Consolidation may be complete in a few hours, but usually takes from 12 to 24. The hepatization period varies from three days to two weeks or more, from three to nine days being the usual period. The process of resolution is wonderfully rapid, and with its coming the whole complexion of the case changes in a few hours.

Rapid changes.

Points of Importance.—The morbid anatomy makes clear the physical signs of the disease, but one should remember that areas contiguous to consolidation are hyperæmic and edematous and that bronchitis and pleurisy are invariable accompaniments of fully developed lobar pneumonia. Pleuritic effusion is comparatively slight and usually undiscoverable by physical examination.

Associated conditions.

Predisposing Factors.—60 or 70% of all cases occur in males and in the United States the colored people suffer more than the whites. Children between the ages of two and six are especially susceptible and after an interval of relative immunity its incidence and mortality steadily increase from puberty to old age. Hence in incidence and mortality it is a disease of the extremes of life. It is most prevalent in the late winter and early spring, is present in all civilized countries whatever their latitude or climatic conditions, and is peculiarly fatal in some high altitude regions, as in Butte, Mont., where the elevation and smelter fumes make it exceptionally deadly. Alcoholism, acute or chronic, is a marked predisposing factor unfavorably affecting prognosis and many acute "drunks" are brought to the police station suffering from an unrecognized pneumonia. Fatigue and exposure to cold and wet if coincident are important factors, one attack invites others and an astonishing number of recurrences have been reported in the same individual. Cold in itself must be regarded merely as a means of depressing vitality and lowering resistance and is more potent when combined with dampness. Coryza and bronchitis often precede an attack, and no doubt furnish a favorable soil for the development of

Males chiefly.

Age.

Season and altitude.

Habits.

Fatigue and exposure

Antecedent disease.

the diplococci. Chronic disease may play a prominent part, and pneumonia is a frequent terminal event in acute or arrested pulmonary tuberculosis, chronic nephritis, diabetes, carcinoma, arterio-sclerosis, prostatic hypertrophy and chronic heart disease.

Complicating.

Varieties of Pneumonia.—*Pneumonia may complicate any acute infectious disease* and is especially frequent in influenza. *The term "typhoid" pneumonia should be confined to pneumonia with an overwhelming toxæmia which produces a typhoid state, not to pneumonia complicating typhoid fever.*

"Typhoid."

Traumatic pneumonia is rare, *migratory pneumonias* both unusual and interesting. *Massive pneumonias* are sometimes encountered in which the large bronchi are plugged and the physical signs greatly obscured by suppression of auscultatory phenomena. *Afebrile pneumonia occurs especially in the aged,* and it should be remembered that *senile pneumonias may lack chill, high fever, cough, crisis, expectoration and typical physical signs. Central pneumonias* are very misleading, and it may be days before the process reaches the surface and yields physical signs of a positive nature.

Traumatic, migratory, massive.

Afebrile and senile.

Central.

Delayed Resolution.—In the author's experience *long delayed* resolution is an extremely rare event, usually proving to be an empyema; but slow resolution is common, particularly in the lobar pneumonia of children.

Selective Points.—The right lung is affected in about 60%, the left in 30%, and both in from 10 to 15% of all cases. As regards the lobes affected, apex pneumonia occurs in about $\frac{1}{5}$ of the cases and the whole lung is affected about as often as is the lower lobe alone, in which the inflammation usually commences.

General Comment.—In these United States from 70,000 to 80,000 of our people die each year of lobar pneumonia, and the disease is apparently increasing. As Osler says, it is the real "Captain of the Men of Death," and the true "friend of the aged." Its apparent increase may be due to more careful methods of diagnosis, but it is probable that other factors are operative.

Terrible mortality.

Increasing.

Symptoms.—The ordinary symptoms of pneumonia are (a). *Chilliness* or repeated rigors, for which convulsions may be substituted in children. (b). *Fever.* Rising abruptly from normal to 103–105° F. and tending to assume a continuous form, it lasts throughout the period of congestion and hepatization (ordinarily from 3 to 14 days) and terminates by crisis. (c). *Crisis.* A critical drop in temperature and the dramatic contrast between intense suffering and relative comfort is an

Striking usually.

astonishing phenomenon. The fall usually reaches subnormal and is attended by sweating or perhaps diarrhœa. Immediately before it there may be a marked rise in temperature (pre-critical); following it there may be a secondary elevation rapidly subsiding to normal (post-critical). *A pseudo crisis* may occur and raise false hopes, and finally, *an ante-mortem rise or fall* may occur. The latter is most misleading, but is ordinarily associated with other signs of impending death that should prevent misconstruction. (d). *Pain* is almost invariably present and *is distinctly pleuritic in type.* (e). *Cough and Expectoration.* In the early stages the cough is dry, painful and unproductive, the sputum is at first scant and mucoid, then muco-purulent and blood-streaked, later, during hepatization, it becomes rusty and during resolution creamy, in appearance. The aptly named "prune juice" sputum is of serious portent and the sputum of the first two stages is so extraordinarily tenacious as to cling to the lips and to the bottom of the inverted sputum cup. *Respiration.* Dyspnœa is always present, the respiration varying from 30 to 70 per minute, according to the extent of involvement and still more to the severity of the toxæmia, and the pleuritic pain causes a grunting respiration and shallow breathing. *Heart and Pulse.* The pulse is full and bounding, *but as in typhoid, relatively slow.* The pulse-temperature ratio and the condition of the heart sounds are important factors in prognosis. *If the heart is acting properly the pulmonary second sound is strongly accentuated and its marked diminution or absence points to right sided cardiac dilatation and impending failure.**

Critical signs.

Rusty sputum.

Prune juice sputum.

Grunting.

Prognostic signs.

The Blood.—In pneumonia *leucocytosis* roughly measures the reciprocal action of toxæmia and resistance and its absence is ominous. The average number of leucocytes is about 25,000 per cm., and counts between 30,000 and 50,000 are not uncommon. The significance of a failure of leucocytosis is somewhat diminished in complicating pneumonias such as those associated with typhoid or smallpox, diseases in which the vitality of the patient may be greatly lowered. *The leucocyte count ordinarily reaches normal within two or three days after crisis and its undue persistance suggests septic complications. The Urine.* Albuminuria is common but is usually of the febrile type subsiding promptly with the termination of the active process. *The urinary*

Leucocytosis the rule.

Persistent leucocytosis.

* From clinical experience the author would advise special care in the lobar pneumonia of arrested or apparently cured tuberculous cases. Many of these experience sudden collapse after a few days' illness and often at the time of apparent crisis.

Albumin and chlorides.

chlorides are diminished or absent during the active stage of the disease and the urine is scant, dark, and of high specific gravity.

Meteorism.

Gastro-intestinal Tract.—The tongue is ordinarily furred, vomiting or diarrhœa may be present, and meteorism may greatly embarrass circulation and respiration.

Headache and Delirium.—Severe headache may be present at the onset or more rarely throughout the patient's illness. Delirium is unusual, the patient being ordinarily rational and acutely sensible of his situation and either low or active delirium is ominous. Maniacal delirium occasionally occurs, and delirium tremens frequently develops in alcoholic cases.*

Physiognomy of Pneumonia.—If the patient be conscious, the expression is anxious, eye bright, the attitude active, and the decubitus usually lateral, the affected side being most often undermost during the early stages. Herpes appear on the lips in a majority of cases and dyspnœa is evident both in the chest movement and the working nostrils. Both cheeks may be flushed, or that of the affected side alone and more or less cyanosis is always present. Deepening cyanosis should be carefully watched for and met by appropriate treatment directed to the circulation.

Herpes.

Alæ nasi.

Filush.

Physical Signs.—Inspection.—Aside from the physiognomy and decubitus one notes *lagging* or *immobility* of the *affected side* as a whole or chiefly over the lower zone if the base only be involved, increased rate of respiration and overaction of the accessory muscles of respiration.

Lagging.

Palpation.—Confirms inspection and if *fremitus* is tested *after cough* (to remove obstructive secretion) *it will be increased* unless the larger bronchi are filled with fibrinous exudate (massive pneumonia). Pleural friction may be felt in some cases and palpatory percussion reveals marked resistance over the affected area.

Increased fremitus.

Percussion.—*Dulness with well marked resistance* is the sign of *consolidation, hyperresonance or tympany* that of *engorgement*, and in early or doubtful cases percussion should be made while the patient is lying prone, or, if deemed safe, sitting up and leaning forward to avoid confusing differences due to the unequal thoracic pressure involved in the lateral position. Percussion may be wholly negative in central pneumonia for several days but is so frankly dull, ordinarily (when hepatization is established), as to require no precautions as to posture and leave no doubt in the mind of the examiner. Equally clear is the

Confusing factors.

* Too frequently the pneumonia is overlooked in such cases.

hyperresonance above the dull area (advancing infiltration—vicarious emphysema—impaired tonus) which *may even simulate the cavity percussion note* in quality and *variations*, especially in cases of apex pneumonia. Hyperresonance.

Auscultation.—In the *first stage*, or *in massive pneumonia, the breath sounds may be suppressed* and one should seek to hear the true crepitant râle at the end of inspiration. *As the consolidation advances and nears the surface the intensity and bronchial character of the sounds increase pari passu until exquisite, dry, tubular breathing is audible* over the *area of consolidation* however varied may be the râles over adjacent areas or in the opposite lung. Value of crepitation.

The *voice sounds may* be normal or diminished early, but, as a rule, *give better and prompter news* of an advancing process than do the breath sounds, and ultimately yield the pure broncophony of superficial consolidation. As in palpation, if the signs are obscure, the patient should cough to clear the bronchi before auscultation is completed. In the *stage of resolution* there is a rapid recession of the pure tubular breathing and voice conduction, associated with the reappearance of fine crepitation, shortly succeeded by coarser moist râles, together with diminishing percussion dulness and fremitus. Voice sounds best guide.

Diagnosis.—Diagnosis depends upon the *frank, febrile* onset and continuous high fever, pulse temperature ratio, rusty sputum and physical signs, and the sputum is often characteristic when physical signs are lacking (central cases). The only confusing conditions are *afebrile senile pneumonias* in which the physical signs and toxæmic symptoms must govern; *cases of pleural effusion with transmitted tubular breathing*, differentiated by the flatter percussion note, displacement of the heart, more distant quality of the tubular breathing and relatively slight fremitus, *and, positively*, by the exploratory hypodermic puncture and removal of the fluid; *cases of acute pneumonic tuberculosis*, in which an existing or pre-existent lesion or a tainted family history may be suggestive, but which can often be differentiated only *by the subsequent course*, evidences of a destructive process and the recovery of the tubercle bacillus from the sputum; *hypostatic pneumonia occurring in a febrile disease*, in which case the primary condition itself usually suggests the probability of hypostasis which is often incomplete, usually bilateral, markedly and persistently dorsal, and in correspondence with and dependent upon the decubitus of the patient and heart strength. Usually easy. Misleading conditions. Exploratory puncture. Hypostasis.

Broncho-pneumonia is wholly different as to course and sputa,

Usually unlike.

and in physical signs, save in the exceptional cases of extensive fusion of broncho-pneumonic areas, and in these the course and associated and antecedent pulmonary signs are sufficiently distinctive. *Infarct* is usually suggested by the primary disease with which it is associated, its *instantaneous onset and limited area*, bright bloody sputum or actual hemoptysis.

Finally.—*Central pneumonia* which *may* not be positively diagnosed until rusty sputum appears or the signs become evident from superficial consolidation, but as a matter of fact usually *is* recognized at least tentatively before these evidences appear, even in its larval forms, by its *onset* and the *suggestive* though not clean-cut *localized departures from normal physical signs*.

Usually detected.

Prognosis.—Pneumonia is an extremely fatal disease and kills from 10 to 70 or even 80% of the cases affected, according to the virulence of the type and the age and vitality of the individual. *The outlook is bad at the extremes of life, in the obese, in those of bad habits and in such as are debilitated by overwork, unfortunate sanitary environment or chronic disease.* The tuberculous experience a high mortality, and recurrent cases are somewhat more dangerous than primary attacks. Influenzal pneumonia of the lobar type is especially fatal though sometimes peculiarly transient. *Excessive rapidity and arrhythmia both as affecting pulse and respiration are ominous signs, as also are extreme cyanosis, absent or markedly diminished pulmonary second sound, profound albuminuria, low leucocyte counts, and marked delirium.* In a high class private practice at least 10% of all cases die, in general hospital practice the mortality will vary from 20 to 40%. In patients above 60 years of age $\frac{2}{3}$ will die.* The essential element in prognosis and treatment consists in the appreciation of the fact that one is dealing with a toxæmia to which all local manifestations are secondary, and that hope lies in diminishing its virulence, and increasing the natural resisting power of the patient.

Important factors.

Heart and respiration.

Essential element.

BRONCHO-PNEUMONIA.— (catarrhal pneumonia, lobular pneumonia, capillary bronchitis.)

Definition.—*An acute mixed infection associated with profound toxæmia and an inflammation having its seat primarily in the terminal bronchioles and lobular tissue of the lung.*

Etiology.—No special germ has been identified, but the diplococcus

*So many variations enter into both private and public practice that arbitrary figures become ridiculous and our judgment must depend upon a knowledge of the favorable or unfavorable factors in each case.

pneumoniæ is most often found, usually in connection with other germs, such as the pyogenic streptococci, Friedlander's bacillus and more rarely Klebs-Lœffler and influenza bacilli. The general predisposing factors are essentially the same as in lobar pneumonia (see p. 139). 75% of all pneumonias in children are of this type and of these about ⅔ follow bronchitis. It is very common in the aged and as a complication of acute and chronic disease. Any irritant, as irritating fumes or dust, the vapor of anæsthetics or the aspiration of food particles may cause it in the presence of pathogenic germs.

Mixed infection.

Morbid Anatomy.—The first stage is one of intense congestion and the capillary bronchiolitis and alveolar congestion is followed by a serofibrinous or hemorrhagic exudation associated with pronounced desquamation of epithelium. Ordinarily an antecedent bronchitis extends to these structures, the bronchioles become plugged; the alveoli gradually lose their contained air and collapse. The exudate, ordinarily termed catarrhal, is sometimes distinctly fibrinous but its cellular constituents show a predominance of epithelium over leucocytes. The process is bilateral, affecting chiefly the lower lobes posteriorly; the lung appears mottled with bluish brown airless depressions indicating collapsed areas, interspersed with bright red emphysematous and crepitant fields. Fusion of areas may bring about extensive consolidation or the affected portions may remain isolated and are sometimes so minute as to constitute an *acute miliary broncho-pneumonia*. Collapsed areas may be distended by the blow pipe, the lungs are larger than normal, have a nodular feel, and drip on section and the process of resolution is much the same as in lobar pneumonia, but is unfortunately more irregular in its points of development so that the disease ordinarily terminates in protracted lysis.

Bronchiolitis.

Catarrhal exudate.

Bilateral.

Fusion of areas

Miliary form.

Fig. 50.—Diplococcus Pneumoniæ. (Frankel-Weichselbaum) and the pneumo-bacillus of Friedlaner. Upper segment shows former, lower segment shows latter.

Two Chief Divisions of Broncho-pneumonia.—The disease may be divided into primary and secondary cases. The former come on acutely and terminate as a rule quite abruptly, the latter are invariably subsequent to a bronchitis or complicate some existing disease. The primary type is infrequent in patients over 4 years of age but constitutes about ⅓ of the cases in children. The primary congestive form may kill in from 12 to 24 hours or may terminate favorably in a few days. The secondary form lasts for weeks.

Primary vs. Secondary.

Sub-divisions.—Aside from the general division into primary and secondary cases, we recognize three forms of disease, (1). An *acute congestive broncho-pneumonia*. (2). *Disseminated broncho-pneumonia*, often described as capillary bronchitis, and (3). The *common form** of the disease. 90% of forms (1) and (3) are bilateral, the disseminated form invariably so. In the acute congestive and the common form, the signs are always most marked posteriorly and nearly always in the interscapular spaces. Save in the acute congestive pneumonias, the symptoms and physical signs are *strikingly pulmonary* from the outset, though sometimes obscure, confusing, and out of accord with the degree of involvement shown by autopsy in fatal cases. The nature of the pathologic process explains this fact by showing that even where consolidation exists the condition of the surrounding tissues may be such as to produce a hyperresonance which masks the percussion signs of consolidation.

Signs posterior.

Pulmonary at onset.

Symptoms of the Acute Congestive Form.—This may be rapidly fatal, killing in 24 or 48 hours and the physical signs may be limited to harsh breath sounds and possibly a slightly impaired percussion note. The signs are usually bilateral but not necessarily of equal intensity. *The onset is sudden*, often with vomiting and occasionally with convulsions in children or chill in the adult. *The fever* runs *high* and may steadily increase to a fatal termination (103–107). *Cough may be entirely absent or violent and harassing. Pulse and respiration are extremely rapid, cyanosis marked, mental dulness and apathy early and extreme.* It may merge into the common form of the disease or terminate sharply by crisis.

Rapid.

Sudden onset.

Toxæmia profound.

Acute Disseminated Broncho-pneumonia.—This form which some of the foreign clinicians still term "capillary bronchitis" is primarily an inflammation of the capillary bronchi, but, post mortem, is invariably associated with areas of true broncho-pneumonia. Like the other forms *it comes on acutely, pulse and respiration are rapid, prostration* is *marked but not immediate, dyspnœa and cyanosis are prominent, the cough is severe though not sharply painful*, but *the temperature* usually runs *low*. The physical signs consist chiefly of the sub-crepitant and coarser râles, generally distributed, always bilateral, and best marked posteriorly at the bases. *The percussion note is hyperresonant or even tympanitic*, and the disease ordinarily runs a short course of a few days or a week or ten days and terminates by lysis, or merges into the common form of the disease. Recovery is the rule.

Low fever.

Basal signs.

Hyper-resonance.

* Following Holt's classification.

Symptoms of the Common Form.—*The onset is abrupt, fever high but* with rare exceptions strikingly *remittent* (often 3 to 4 degrees), *vomiting is frequent* and *convulsions in children a rare initial event. Cough is early, intractable and persistent, harassing but not usually painful. The respiration is rapid* (60–80 or even 120 in severe cases) *and may be noted as a premonitory symptom even before the frank onset; prostration is marked and shortly becomes extreme; expectoration is absent in children, cyanosis* becomes *marked in all severe cases,* and *late convulsions* may occur as one of the terminal events. In rare instances infants or badly nourished children may show a low temperature. The pulse may reach 200 and *is always rapid* and sometimes irregular, the signs of *actual dyspnœa are pronounced,* the alæ nasi being active and inspiratory recession of the lower interspaces marked. *Gastro-intestinal disturbances* may be present and greatly complicate the treatment, flatulence and exhausting diarrhœa or persistent vomiting aggravated by cough being the most troublesome factors. The urine is febrile, being scanty, high colored, containing excess of urates and perhaps a trace of albumin. The ordinary course is from two to three weeks and it usually terminates by lysis. Cases may however be prolonged for two or three months or a chronic broncho-pneumonia may succeed the acute form and often develops into a tuberculosis.

Early rapid respiration.

Rapid pulse.

Dyspnœa.

Gastro-intestinal symptoms.

Termination.

Physical Signs.—These are primarily *the signs of an acute bronchiolitis,* the most characteristic sign being the subcrepitant râle heard chiefly and predominantly over the lower lobes in the interscapular spaces. Unlike lobar pneumonia the left lobe is usually chiefly affected though both are involved. *Bronchial râles,* dry and moist, of a larger and coarser character *may mask the finer crackles* and after a variable period *evidences of consolidation may appear* over scattered areas or more rarely produce extensive consolidation.

Left lobe chiefly.

Inspection.—Inspection reveals the *facies of profound illness* and its pulmonary type is indicated by the evident *dyspnœa, harassing cough,* slight or marked *cyanosis* and *inspiratory recession of the lower interspaces. Palpation* is *negative in the congestive type, fremitus* is *diminished in the disseminated but may be increased in the "common form"* if consolidation be sufficiently extensive. *Percussion* is *negative in the congestive form or shows only slight dulness,* there is *hyperresonance or tympany in the disseminated variety,* while *in the common form scattered areas of impaired resonance may alternate with hyperresonance.* Furthermore, certain areas may disappear and reappear from day to day,

Too sick for bronchitis.

Vague signs.

Use light percussion.

or hour to hour. Light percussion should invariably be practised and it should be remembered that consolidation areas are first and chiefly evident in the interscapular spaces save in those instances, usually tubercular, in which the localization is anterior and apical. *Auscultation* is by far the most valuable procedure. *The characteristic subcrepitant râles are evident, overshadowed perhaps by their coarser brethren, and areas of increased voice conduction and harsh or even tubular breathing may make consolidation manifest to the ear even though it is not to the percussing finger.* Friction sounds are rare and it should never be forgotten that *all signs may be lacking save in forced deep inspiration, often obtainable in children only through the act of crying*, and furthermore, that *in all cases of broncho-pneumonia the patient is too sick for a simple bronchitis which the disease may simulate in its early stage or may directly follow.*

Most valuable procedure.

Crying assists.

Secondary broncho-pneumonia is a common event in certain of the acute exanthemata, especially measles, scarlet fever and diphtheria, and as a terminal event in cachexias and senile conditions. In the latter, its course may be insidious and lacking in the franker clinical manifestations. *In measles and scarlet fever* it is severe and protracted, in *diphtheria* extremely fatal. In influenza one meets with two types, one extremely brief, the other protracted and severe. The latter form is peculiarly liable to leave behind it a persistent spasmodic cough.

Serious complications.

Persistent cough.

Complications.—Ileo-colitis is dangerous and troublesome when it occurs. Meningitis and nephritis are rare, tuberculosis is not uncommon and is undoubtedly often primary. Endocarditis is fortunately a clinical curiosity. Suppuration and abscess formation are rarely seen save in aspiration and deglutition cases.

Termination.—Many cases are prolonged for months with recurring attacks and some ultimately recover though many prove tuberculous and run the course of a chronic tuberculosis or of fibroid phthisis.

Tuberculosis.

ATELECTASIS.—Definition.—A condition characterized by localized or extensive, temporary or persistent, loss of the pulmonary air content.

Etiology.—Aside from atelectasis in the new-born child resulting from extreme weakness or actual obstruction from meconium or mucous, the condition is essentially secondary to obstruction of the bronchi, large or small, i.e. broncho-pneumonia, obstruction by tumors, membrane, foreign bodies, or the pressure of growths or exudates, i.e. mediastinal or pleural growths, pleural effusion, pneumothorax, greatly enlarged heart, pericardial effusion, etc. Slight grades result from mere deficient

Usually secondary and subordinate.

expansion, as in adenoids, enlarged tonsils or in delicate women and children. In extreme cases the lung is carnified and persistent atelectasis leads to atrophy or fibrosis. Adenoids and the like.

Diagnosis.—*The presence of an adequate cause*, inspiratory recession of interspaces or actual retraction, absent or distant tubular breath sounds and corresponding fremitus make the condition clear. These are for the most part silent or hushed areas though in cases of bronchopneumonia deep inspiration may convert them into noisy ones. The condition is so generally secondary and subordinate as to require no extended notice here.

CONGESTION OF THE LUNGS.—Active congestion and the three forms of passive congestion which include so-called pulmonary edema will be discussed under this head.

Active Congestion.—The inhalation of irritating substances chiefly vapors and gases and the primary stage of pneumonia are the chief factors in the production of an extensive, active congestion and it is so generally but a stage in a dominating disease process as hardly to deserve extended description. (See lobar pneumonia, p. 138.)

Passive Congestion.—Sharply drawn lines between obstructive and hypostatic congestion and between these and pulmonary edema are unfortunately largely confined to the autopsy room. Between the two former there is little need of distinction for both arise from an obstructed circulation whether from valvular disease, a weakened myocardium, toxæmia, the pressure of tumors, pleuritic or pericardial exudate or abdominal distention, or from cerebral lesions. Clinically *the hypostatic form* as opposed to the simple obstructive congestion *is encountered in conditions of profound exhaustion* especially such as are characterized by the typhoid state, i.e. it represents profound æsthenia. *Edema accompanying acute congestion or inflammation is termed inflammatory as opposed to the ordinary edema of the lungs associated with passive congestion.* This latter form is a terminal event in many diseases and may come suddenly and without warning or supervene upon prolonged congestion. In certain cases it seems to be purely toxæmic but in general its etiology is that of the associated congestion. Dubious distinction

Morbid Anatomy.—Long continued passive congestion may produce brown induration, the organ being dense, bulky and russet brown; its capillaries are distended, the bronchioles and alveoli filled with epithelium, blood cells and pigment, and there is connective tissue increase. This represents especially the obstructive congestion typified in

incompensated mitral lesions. In the *hypostatic form* the engorgement is intense and portions of the lung sometimes airless. If these areas are extensive the term *splenization* is applicable. *In edema* the bulky lung is sodden and the foamy yellow or reddish serum infiltrates the interstitial tissue and fills the air passages. Any of the forms of passive congestion may be predominantly unilateral *but are usually bilateral.*

Seek cause.

Diagnosis.—The existence of a cause is the first link in the dubious chain. The symptoms of acute congestion of the severer form are described under pneumonia. The milder types of all varieties may produce but slight subjective and objective symptoms. In cardiac disease for example the condition may not attract notice until there is marked dyspnœa and a blood streaked sputum containing the peculiar pigment bearing cells. A careless practitioner often entirely overlooks *hypostasis* until it has reached a pneumonic stage (splenization). Less frequently mild forms of edema occur which may be transitory. In the severer forms of passive congestion dyspnœa and cyanosis are present and in marked edema are excessive. In all there is *restriction of chest movement pari passu with the area involved. Fremitus* may be diminished or increased according to the degree of actual consolidation present. *The percussion note may be hyperresonant from relaxation or dull from consolidation. Auscultation may reveal harsh, diminished, absent or tubular breathing according to the stage of congestion, the presence of atelectasis or, true consolidation. Râles,* both dry and moist are present, their pitch and quality varying with the pathologic conditions. *In edema the subjective symptoms are especially pronounced,* the sputa profuse, frothy, serous or blood stained and in inflammatory edema present the "prune juice" appearance. In edema, moreover, the râles are more liquid, the breath sounds and voice conduction suppressed or feeble. Slight crepitation at a base in a nephritic subject, or any patient suffering from the diseases associated with passive congestion, demands serious attention. Astonishingly sharp congestions may accompany obscure apoplexies unassociated with paralysis or loss of consciousness.* The effect of cardiac stimulation or, in hypostasis, mere attention to the necessary shifting of a patient's position may rapidly and markedly diminish the involved areas, the

Slight symptoms.

Variable physical signs.

Marked subjective and objective signs.

Test manœuvres.

* In a case recently observed two cerebral hemorrhages of the silent type occurred without loss of consciousness or power, the only symptoms being sudden pallor, mental confusion and in the second attack transient motor aphasia. In each there was marked pulmonary congestion lasting for about 12 hours. A third hemorrhage ten days later produced almost instant death.

latter condition being often due to one fixed posture in senile, obese, cachectic or profoundly toxæmic patients.

TUBERCULOSIS. (Pulmonary phthisis, consumption).—**Definition.**—*An infectious disease caused by the tubercle bacillus of Koch, usually chronic, rarely acute, characterized by the formation of tubercles or a diffuse infiltration of tuberculous tissue and tending to ulceration, fibrosis or calcification.*

Distribution.—It is a universal disease visiting practically all races and every latitude. In the United States alone it kills at least 150,000 persons each year, and the annual loss in potential wealth has been estimated at $500,000,000. It costs the life insurance companies of America at least $6,000,000 annually, and as the deaths occur chiefly at early ages when few premiums have been paid, it constitutes the source of their greatest early loss. Domestic animals and pets, such as cattle, pigs, guinea pigs, rabbits and monkeys are extremely susceptible; horses, dogs, goats and cats less so. Cold, damp, densely populated districts suffer most, and dry and high altitude regions least.

An unnecessary scourge.

Susceptibility.

Morbid Anatomy.—If a pure culture of tubercle bacilli be injected in the tissues of a susceptible animal it produces tuberculosis. From the tuberculous tissues the germ can be recovered, grown in pure culture, again used for inoculation and so on indefinitely. This absolute proof of its infectious nature makes the position of those fighting the "great white plague" unassailable. *Changes Subsequent to Inoculation.* (a). Grouping of germs in tissue by multiplication and their distribution by the lymph current. (b). The formation of epithelioid cells, about five days after inoculation, by multiplication and by metamorphosis of the cells of the capillary endothelium and connective tissue. (c). Outwandering of leucocytes to focus of infection and multiplication of mononuclear forms. (d). The formation of a marginal reticulum of connective tissue. (e). The production of large epithelioid cells containing from four to twenty nuclei grouped at the poles or periphery. These *giant cells* are most common in lupus and in glandular and bone tuberculosis and the more chronic and slowly developed the process, the greater is their number. In acute lesions they are scant and absent, nor are they peculiar to tuberculosis, being found in other of the granulomata such as syphilis. *Caseation and Sclerosis.* The changes described tend inevitably to devitalize the affected tissue and produce areas of central coagulation necrosis converting all structures into a cheesy homogeneous substance. This may be extruded, leaving cavities of greater or lesser size, or, more rarely, calcification may accom-

Cause proven.

pany encapsulation. Many cases terminate spontaneously without reaching caseation and often without yielding any recognizable symptoms.

Slow vs. Rapid.

The Spread of Infection.—It will be readily understood that lymph distribution means a slow infection and infection through the blood stream a rapidly developing and widely disseminated process.

Etiology.—*Race.* The Hebrews are to a certain extent immune, but in this country the Irish and the Scandinavian, the Indian and the Negro show a heavy mortality. *Sex.* Its incidence is about equal in the two sexes. *Age.* Children are peculiarly prone to affections of the glands and bones. After the age of childhood the lungs are chiefly affected and susceptibility diminishes considerably at the age of 30. *Occupation.* Occupations involving exposure to extremes in temperature, dust and dampness are distinctly unfavorable. Workers grinding glass or steel or handling furs show a high mortality. *Sanitary Conditions.* Lack of sunshine, fresh air, overcrowding, filth and physical exhaustion are all predisposing factors. Amongst the tenement classes all conditions are favorable to its development and transmission. *Pre-existing Diseases.* Tuberculosis frequently follows chronic disease of the tonsils, adenoids, neglected colds, broncho-pneumonia, lobar pneumonia, pleurisy, influenza, measles, whooping cough and entero-colitis, and may complicate diabetes, aneurism, heart disease, locomotor ataxia, sclerosis of the liver or chronic Bright's disease, any condition in short reducing vitality and resistance or tending to prevent free lung expansion. *Injuries.* Tuberculosis of the bones and joints and especially of the vertebra and hip joints in children is frequently ascribed to injury though due no doubt to a pre-existing latent infection. *Heredity.* Direct transmission in utero is probably extremely rare, and heredity is but another name for post-partum implantation of germs upon a fallow soil. The tuberculous mother readily transmits germs to her offspring and a neglect of sanitary precautions in any infected house is peculiarly dangerous to the children. The germs may remain latent in the glandular system for an indefinite period, a fact undoubtedly explaining the increased adult mortality from tuberculosis in families showing the taint. *Individual Predisposition.* The "habitus phthisicus" has long been recognized, certain individuals being from birth less resistant to the germ than are others. Small bones, a delicate complexion, and a contracted chest, long sweeping lashes, large lustrous eyes and silky hair are found in one type; a muddy complexion with marked tendency to glandular swelling, acne and weak eyes mark

Common factors.

Diminished resistance.

Means post-partum infection.

Often latent.

Known types.

the other type. Extraordinary beauty, unusual intelligence and a quick wit were recognized even by the ancients as qualities often associated with tuberculous predisposition. The children of either type represent undoubtedly cases of latent infection and need unusual care along hygienic lines. Having this, they may grow up to manhood strong and well, and die of old age. May escape disease.

Preferential Site of Lesions.—Almost any portion of the body may be involved, the lungs most frequently in adults, the bones, lymph glands and intestines in children, while the peritoneum, kidneys or brain may be involved at any age. Secondary involvement of the intestines is very common in advanced pulmonary tuberculosis.

Modes of Onset.—It should never be forgotten that *nearly every case of tuberculosis, whatever its form, gives a history of previous impairment of health.* As regards actual onset, it may be acute miliary and widespread, or wholly glandular, pleuritic or pneumonic. Previous health.

ACUTE MILIARY TUBERCULOSIS.—(Acute tuberculosis. General diffuse tuberculosis. Acute disseminated tuberculosis.)

Cause.—Introduction of tubercle bacilli, from a latent or active glandular, osseous, or pulmonary focus, into the *blood stream.*

Morbid Anatomy.—Viscera show general changes of an acute febrile infection and a general distribution of miliary tubercles, the lesions in the lung, pleura or brain predominating and the peritoneum or more often its diaphragmatic surface being frequently affected.

Symptoms.—*Typhoidal Form.* The onset may be gradual and exactly simulate typhoid. The fever rises gradually or more rarely, sharply, and to a variable degree, 103–105° F. being common and afebrile cases rare. The symptoms may be those of the "typhoid state" but the following are prominent and should be carefully noted: —(a). *Markedly accelerated breathing.* (b). *Subjective and objective dyspnœa.* (c). *A rapid weak pulse.* (d). *Cyanosis.* (e). *Sweats.* (f). *A variable, often intermittent or inverse temperature.* A *continuous* temperature may be present for some days prior to the development of *intermittency.* Usually but not invariably one observes (g). *cough* unproductive or attended by purulent or muco-purulent sputum. (h). *Tubercle bacilli, which may be present* early, more often late, or, in rare instances, be absent throughout. (i). *Herpes labialis.* (j). *Rapid emaciation* is constant. Important signs. Bacilli may be absent.

Physical Signs.—At first they are absent or only bronchitic in character and for days or weeks may be scant or misleading. A careful examination may demonstrate the presence of *ante-* Usually scant.

Hyperresonance.

cedent tuberculous lesions. The author believes that hyperresonance is the most constant and significant early sign. In rare instances patches of consolidation are clearly defined. *The enlargement of the spleen occurs much later than in typhoid, and the same may be said of Ehrlich's diazo-reaction. Widal's test* is absent, and the stools are quite unlike those of typhoid. Furthermore the pulmonary symptoms are strikingly predominant even in that acute general form which presents abdominal symptoms.*

Course.—Six to twelve weeks. **Prognosis.**—Almost invariably fatal, though rarely the acute symptoms subside leaving a progressive but limited lesion. **Comment.**—The general symptomatology of the disease is that of an acute intoxication with disseminated miliary foci and these facts explain the absence of marked physical signs. Cases may be wholly pulmonary or predominantly abdominal, but a division into pulmonary, typhoidal and peritoneal is useless.

Usually broncho-pneumonic.

ACUTE PNEUMONIC TUBERCULOSIS.—(Phthisis florida, galloping consumption, acute tuberculo-pneumonic phthisis). This is a *relatively* rare disease representing not over 2% of the cases of pulmonary tuberculosis. It is usually broncho-pnemonic in form; rarely, lobar.

The lobar form may exactly simulate an ordinary pneumonia, *but one finds ordinarily a history of previous illness or signs of old lesions.*

Early diagnosis difficult.

In these cases the destructive process is often rapid and extreme and the author recalls one in which at autopsy the right lung was found to be entirely destroyed, leaving a huge cavity; the left infiltrated and hypertrophied to such an extent as to force the heart to the right and produce a concentric dexiocardia. Instead of a true crisis, septic temperature develops with sweats and a purulent sputum containing tubercle bacilli but in some instances the process subsides, becomes chronic and may even become arrested. *Early positive diagnosis is usually impossible.*

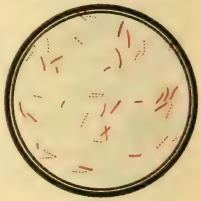


Fig. 51.—Tubercle bacilli in sputum.

Later, is evident.

Bacilli often present early.

The broncho-pneumonic form, is characterized pathologically by broncho-pneumonic lesions and shows a tendency to fusion, caseation and cavity formation. It presents the symptoms of an *acute broncho-pneumonia followed by the signs of septic absorption, pulmonary infiltra-*

* As a matter of fact the confusion of typhoid fever and miliary tuberculosis need but rarely occur even if the diagnosis lack the assistance of the Widal test.

tion and cavity formation. Tubercle bacilli may be and usually are present early but their appearance *may* be delayed. Hemoptysis may occur and is sometimes the first event.

Comment.—In both the lobar and broncho-pneumonic forms of acute tuberculosis a delayed diagnosis is almost always necessary, the most careful questioning as to previous health and family history may yield no information, and the appearance of tubercle bacilli and the signs of destructive infiltration associated with hectic may be the only means of diagnosis.

CHRONIC ULCERATIVE TUBERCULOSIS.—Definition.—A tuberculous disease of the lung characterized by chronicity, variability in severity and frequent intermissions. Aside from individual resistance, its termination in recovery or death depends upon the promptness with which a diagnosis is made and rational measures instituted. The *sine qua non.*

Pathologic Anatomy.—The lesions are ordinarily those of a chronic tuberculous broncho-pneumonia; the terminal bronchioles and the alveoli being the seat of an inflammation forming areas of peri-bronchial pneumonia; subsequent changes depending upon the activity of the process and the resistance of the individual. Usually a fusion of areas results in ulceration and cavity formation. Favorable cases result in fibrosis, capsular limitation and arrest more often than is ordinarily supposed, spontaneous recovery from *unrecognized* tuberculosis being common, as shown by the post mortem records at home and abroad, contracted apices, peri-bronchial nodules and adhesions being frequently found. Inflammation of lung tissue in this disease leads to pleuritis and limitation of lung movement. The areas involved furnish the signs of broncho-pneumonia either sharply localized or disseminated, but large tracts may become involved with a corresponding increase in the frankness of the pulmonary symptoms. By necrosis of the bronchial walls through inflammation aided by the pressure of retained secretion and a destructive ulceration, cavities are formed, the physical signs of which are fully discussed on page 90. New cavities without a firm limiting membrane may yield doubtful signs, particularly if they occur in the center of a caseous area lacking bronchial communication. Vomicæ vary in size, sometimes representing almost the whole right or left chest as in the case mentioned on page 154. Small

Fig. 52.—Various forms of Pulmonary Cavities; Incipient Tubercular Deposits; Area of Softening (Right Inferior).

Fusion of areas.

Fibrosis.

Pleuritis.

Cavity formation

cavities are obliterated by a process of absorption, fibrosis and contraction; large cavities may be partially obliterated by the same processes and become inactive. Hemorrhage may occur at any stage of a chronic ulcerative tuberculosis and though the arteries are usually resistant, *may* be the first symptom. The amount varies from a mere streak of blood to the rare flooding that kills almost instantly. Bronchiectases are common in advanced cases, the intestines are often involved and this complication, like ulceration of the larynx, is painful, exhausting, and tends to greatly shorten life.

Hæmoptysis

Bronchiectasis.

Symptoms.—*Early symptoms.* The modern theory of the curability of tuberculosis depends upon early diagnosis. Every physician knows that cases referred to him as latent or incipient are likely to be far advanced, i.e. in the second or even third stage of the disease. They should be recognized before the stage of extensive infiltration and certainly before softening has begun if the best results are to be obtained. When so recognized at least 80% can be restored to apparent good health, and the larger number of these should remain well under proper conditions. Aside from tubercle bacilli and cavity signs no one symptom is conclusive, but those following should be considered. (a). *Loss of weight. The present weight, the best weight, and the weight prior* to any noticeable impairment of health should be ascertained, as few cases develop without *progressive* weight loss.* (b). *Indigestion.* Dyspeptic symptoms are common in the early stages of tuberculosis, and loss of weight is often attributed to that cause when it should be traced further back. (c). *Cough. This may be entirely absent* and is frequently unnoticed and denied by the patient, even though audible to the examining physician. It is often harassing and troublesome, particularly in the advanced stage of the disease, and may be entirely unproductive, hardly more than a clearing of the throat, or, associated with profuse expectoration or even hæmoptysis being oftentimes bronchiectatic in advanced cases. (d). *Sputum.* The sputum may be absent, mucoid, muco-purulent or purulent, according to the stage of the disease. Tubercle bacilli are present in all advanced cases, but a diagnosis must often be made in their absence. When found by proper staining methods the evidence is positive and final. (e). *Pain* may or may not be present, and is often represented by a feeling of mere oppression, at other times being distinctly pleuritic in type or dull and ill-defined. (f). *Anæmia* is frequently present in the early stages of the disease, and

Early diagnosis.

Curability.

Progressive loss.

Common.

Often denied.

Often absent.

Variable.

Seldom extreme.

* Robust appearing heavy weights may have lost thirty or forty pounds and such patients often receive scant sympathy and no proper examination.

always in advanced cases. In the former it is sometimes of the chlorotic type, but usually one finds a *secondary* anæmia (see pp. 397, 402). (g). *Dyspnœa.* Dyspnœa on exertion is not uncommon early and is an invariable *late* symptom. (h). *Bronchitis.* A diffuse obscurant bronchitis or often spasmodic asthma is sometimes present and may mask the true lesion. In other instances it is a *localized and suggestive bronchitis.* (i). *Pleuro-pericardial friction.* The author has come to regard with great suspicion *frictional murmurs, pleuro-pericardial in type and site, not associated with a definite attack of pericarditis or pleurisy, but with impairment of the general health.* In several instances these have proven the first demonstrable symptoms of a rapidly destructive tuberculous process. (j). *Night sweats.* This troublesome symptom is ordinarily associated with the final stage, but may be encountered at any time in the course of the disease. Such sweats are chilling and exhausting. (k). *Pulse. In connection with other symptoms* the excitability and undue rapidity of the pulse is extremely important. (l). *Temperature.* In advanced tuberculosis, the temperature is simply that of a septic process, being distinctly intermittent or hectic in type and associated with night sweats. When new areas of the lung tissue are invaded, the temperature is likely to be continuous, or only remittent. One is chiefly interested in the variations in *incipient cases* and here one must emphasize the importance of any persistent rise above the normal. *In all tuberculosis cases with fever, the maximum rise is usually in the evening, but we may have an inverse temperature with the evening normal.* Some cases have no temperature, and one under the author's observation a few years ago, went through the various stages of the disease to death without a rise above 99° F.* *In many cases a temperature will be found only after exertion,* and this is a matter of the greatest importance in relation to both diagnosis and treatment. Furthermore, in such cases a wide variation between the morning and evening temperature, though the latter be within normal limits, may be as serious an indication as the abnormal rise. (m). *Fluoroscopy.* The skilful use of the fluoroscope throws much light upon the condition of the lungs in incipient cases and one often finds a marked limitation of lung movement, as measured by the descent of the diaphragm, quite disproportionate to the amount of lung involved. Areas of extensive infiltration show as shadows, and cavities of consider-

Often confusing.

Suggestive types.

May be early.

Suggestive.

All important.

Afebrile cases.

Exertion temperature.

Valuable.

* The case was not observed in its incipiency, but as no temperature was shown during the stage of softening and when the sputum was filled with tubercle bacilli, it is to be presumed that it was absent in the earlier stages.

able size as bright spots. Litten's sign may be used to test the diaphragmatic movement in the absence of the fluoroscope. (n). *Tuberculin.* The use of tuberculin as a diagnostic agent has its advocates

Fig. 53.—Small yet normal heart in a case of apex tuberculosis. A form of heart frequently encountered in those chests of the phthisical type.

and its bitter opponents. It is argued that inasmuch as the symptoms produced are due to the production of congestion in tuberculous areas, every dose subjects the patient to unnecessary risks. On the other hand its advocates claim that the importance of absolute diagnosis

Routine use unwise.

and the rarity of bad results following its use serve as its justification, and both are doubtless right. The author believes that *its routine use is to be condemned.* On the other hand cases occasionally arise where the slight risk involved may be taken in the interest of correct diagnosis. The fact that it is capable of producing a rise of temperature in apparently healthy controls and in certain cases of syphilis makes its findings less positive than might be wished. (o). *Family history.* The family history, as throwing light upon latent infection, predisposition, or the resistance of the patient, should be carefully investigated as regards both incidence and course of cases in brothers and sisters, parents and collaterals.

Fallible.

Physical Signs.—*A diagnosis must often be made in the absence of positive physical signs, a fact readily appreciated if one considers the genesis of the disease.*

Inspection.—The *physiognomy of advanced tuberculosis* is too well known to need a description, the laity recognizing it as readily as the physician. It is merely a composite of emaciation, hectic, exertion dyspnœa, and often, of chest conformation. *Incipient cases have no distinctive physiognomy* and marked anæmia may or may not be present. The chest may show congenital malformation, and *unilateral wasting of muscle in the suprascapular region is of much importance.* The impairment of chest movement may be imperceptible in the incipient stage, though reduced expansion may be shown by the diaphragm phenomenon or the fluoroscope. Some cases present a high color similar to that noted in the mitral disease of young persons and due, apparently, to interference with the pulmonary circulation. Advanced cases show marked impairment of chest movement, and oftentimes localized contraction, abnormal pulsation along the pulmonary-cardiac boundaries, and, the characteristic phthisical chest.

Importance variable.

Palpation.—Palpation may be wholly negative in the incipient stage, though yielding most exquisite signs in the advanced cases where it reveals lack of expansion, inequality of voice transmission, signs of cavity or marked infiltration (see consolidation p. 142 and cavities p. 90).

Often negative.

Percussion.—In the earliest stages percussion may be negative or actually misleading. Any inequality in the percussion notes of the two sides, particularly at the apex, should attract attention. It should be remembered that, *normally*, the note on the left side is less intense but more resonant and lower pitched than that of the right. Hyperresonance at the apex, if unilateral, is a valuable sign of an incipient

Important data.

Hyper-resonance.

process; in the later stage it is of course likely to indicate vicarious emphysema or the presence of cavities. Patches of dulness may be made out, or extensive areas of infiltration, with or without cavity formation, according to the status of the case.

Auscultation.—This is by far the most important procedure. *Unilateral diminution of the breath sounds or of voice conduction may be as significant as an increase in their intensity.* Harsh breathing and the so-called cog-wheel inspiration must be carefully noted and the latter must not be confounded with the inspiration associated with an over-acting heart, such a condition being frequently met with in tuberculous subjects. Undue prolongation of expiration, though it be low pitched, may be a sign of importance and in advanced cases with marked infiltration and cavity formation the breath sounds are frank and characteristic (see page 101). *Râles* may be absent for a considerable period in the incipient stage. One often hears only fine sibilant sounds, most significant if detected at the apex even in the absence of other signs, and particularly so if unilateral. Every patient should be made to cough in order to bring out râles, and deep inspiration following cough may be attended by sibilant, crackling or bubbling râles or by the so-called mucous click, according to the stage of the disease. In advanced cases the râles are of all types as described on page 104.

Unilateral variation.

Cog-wheel breathing.

Cough manœuvre.

Comment.—Certain points are absolutely essential to the proper examination and early diagnosis of tuberculous cases. A knowledge of the sounds produced by the various manœuvres in the normal chest is absolutely necessary. *Unilateral variations are infinitely more important than bilateral ones in the incipient or early cases.* Inasmuch as the disease usually commences at the apex or apices, this region is the most important for the diagnostician. (Both are affected almost coincidently far more often than is generally supposed.) The primary lesion is commonly slightly posterior to the centre and about an inch or an inch and a half below the apex. The disease tends to extend both upward and downward along the interlobar fissure and in front along the inner margin of the upper lobe. *Posterior apical auscultation* is usually more productive than anterior in the early stages of the disease. *The important areas are the apex, anteriorly and posteriorly, the inner lung borders anteriorly,* the *apex of the axillary space, and, the region of the interlobar fissure posteriorly* as roughly indicated by the scapular border when the arm is placed upon the opposite shoulder. The lungs may often be more involved than physical signs would indicate, as any one may prove by the use of the X-Ray and this fact no doubt accounts

Know the normal.

Apex signs.

Line of march.

Posterior vs. Anterior.

X-Ray.

for the errors in prognosis and failure in treatment in cases judged wholly by the physical signs.

Apex Movement.—It is important to employ auscultatory percussion over the apices in order to determine the height at which they stand and the difference in level as between inspiration and expiration. Marked retraction of one apex is an important sign of either an old lesion, or an active and advancing one. The diaphragm phenomenon has already been described (p. 88).

Height important.

PULMONARY INFARCT (pulmonary apoplexy, hemorrhagic infarct, pulmonary embolism).—*All emboli in the pulmonary artery originate in the right heart* or in thrombotic systemic veins, and may be septic or nonseptic. The former occur in pyemia, ulcerative endocarditis, septicæmia and exceptionally severe acute febrile infections; the latter result from the detachment of vegetations from the tricuspid or pulmonary valves or from a remote non-septic thrombus. The result is usually the blocking of a branch of the pulmonary artery which causes a localized pneumonia primarily representing the distribution of the affected vessel and in some instances leading to abscess or gangrene. The area is usually wedge shaped with its base at the periphery.

Associated ailments.

Peculiar area.

Symptoms.—*Tiny infarcts may be wholly symptomless or produce only slight cough, dyspnœa, and perhaps hæmoptysis. If branches of moderate size be involved these symptoms are more pronounced or indeed extreme and if a large artery be affected instant death results.* The sudden onset of such symptoms accompanied by the physical signs of *circumscribed pneumonia or pleuro-pneumonia, usually basal,* in the presence of recognized causative factors makes the diagnosis easy. *If the embolus be septic the case becomes one of pulmonary abscess or gangrene.* If the septic element be absent prognosis is favorable as regards the lung condition. In septic cases it is bad. Multiple infarcts may occur and their location be easily determined as in a case recently observed; and the picture presented, viz, *sudden localized pain, dyspnœa and bloody sputum* is characteristic even though the physical signs be obscure.

Diagnosis easy, or, impossible.

Multiple lesions.

PULMONARY ABSCESS.—The abscesses *may be single or multiple and due to tuberculosis, septicæmia, pyemia, septic emboli, lobar and broncho-pneumonia, malignant endocarditis and indeed to suppurative disease of any organ or structure adjacent or remote.* There is usually an associated empyema with embolic infarction abscesses which are often multiple. Other abscesses may be distinctly localized primarily but tend to extend.

Often obscure.

Symptoms.—*The symptoms are those of sepsis* (see septicæmia and

pyemia) *and the physical signs may be vague or lacking* if central, or simulate an encysted empyema if superficial, unless perforation occurs (most commonly into a bronchus) and is associated with the sudden appearance of a considerable quantity of purulent sputum which then persists with distinct cavity signs. The sputum is foul, contains elastic tissue and often at first or at intervals more or less blood. It will be noted that the symptoms of sepsis apply to all forms; but non-perforating abscess *may* yield no symptoms or only percussion dulness and, compression.

Perforation.

Sputum.

Prognosis.—Embolic abscess is almost invariably fatal and the prognosis is bad in all though their course may be prolonged or recovery achieved, usually through surgical interference.

PULMONARY GANGRENE.—*The etiologic factors are chiefly those of pulmonary abscess. It is a rare complication of diabetes and pneumonia, exceptionally rare in tuberculosis* and varies greatly in extent, being either circumscribed or diffuse and usually but not always affecting the lower lobe. *The involved areas are surrounded by consolidated, congested or edematous areas. An associated empyema is common and pleurisy invariable in the peripheral lesions.* It shows the same tendency to perforate as does abscess, most commonly into a bronchus, more rarely into the pleura or even the pericardium, esophagus, or, through the diaphragm.

Rare.

Lower lobe chiefly.

Perforation.

Symptoms.—*The only symptom differentiating the disease from pulmonary abscess is the peculiarly horrible odor of the sputum,* characteristically gangrenous and simulated by no other condition except pulmonary carcinoma *with gangrene.* Owing to some peculiar fermentative action the sputum contains no elastic tissue.

Odor diagnostic.

Prognosis.—Embolic malignant and diabetic cases invariably die; in others the prognosis is unfavorable but not absolutely hopeless.

PULMONARY TUMORS.—Only the malignant variety need be considered. Primary sarcoma is a clinical curiosity and carcinoma rare, both being usually metastatic. The most common primary focus is of course the mammary gland, less often uterine, gastric, rectal and osseous growths. Pulmonary carcinoma is most common in middle age.

Primary rare.

Symptoms.—The disease is often strikingly symptomless for long periods or baffling and indeterminate, the size and location of the growth being the chief factor; obstinate and violent cough may or may not be present. Sputum may be entirely absent or like prune juice or currant jelly, blood streaked or even purulent and of gangrenous odor, and contains compound granule cells. Involvement of the pleura may

Frank or obscure.

produce severe pain and the dyspnœa usually present in some degree may be strikingly paroxysmal; pressure symptoms may be marked and are often identical with those of aneurism (see page 205). Fever is often noted as the disease advances, profuse hemorrhage occasionally occurs and there may be marked displacement of the heart. Secondary growths are usually suggested by a primary lesion, but primary ones are frequently beyond a positive diagnosis. The X-Ray may prove valuable in such cases and if the growth be accessible and of considerable size it may yield percussion dulness, usually without tubular breathing, and increased fremitus, more rarely, if attached to a large bronchus, both phenomena.

Pressure symptoms.

X-Ray.

Prognosis.—Death invariably results though only after a period of several months.*

DISEASES OF THE BRONCHIAL GLANDS.—The most important of these glands lie in the angle of the tracheal bifurcation about the main bronchi. The smaller glands follow the course of the bronchi lying in the interlobular connective tissue. They must chiefly be considered in connection with the ailments of children or adults, as possible sources of secondary enlargement with or without symptoms. It should also be remembered that gangrene of the lung may involve them and that through dusty occupation, (inhalation of dust) they may become pigmented and somewhat enlarged. The following diseases may affect them:—(a). Severe acute bronchitis. (b). Scarlet fever, measles, whooping cough, typhoid and similar ailments. (c). Broncho and lobar pneumonia. (d) Pulmonary gangrene. (e). Mediastinal, or, by metastasis, remote, malignant growths. (f). Hodgkin's disease and leukæmia (especially the lymphatic form). (g). Pulmonary tuberculosis. (h). Tuberculous or malignant disease of the *abdominal or retro-peritoneal* structures. The greater number of cases in children are due to tuberculosis, the primary source of infection being either gastro-intestinal, tonsillar or bronchial.

Important areas.

Age.

Occupation.

Associated lesions.

Tuberculosis.

Symptoms.—Many enlargements are symptomless, in other cases the effects are those of pressure within the mediastinum combined with physical signs chiefly observable in the interscapular region and

Pressure.

* In the only case of primary sarcoma of the lung observed by the author no positive diagnosis could be made during life, though the ailment was suspected by reason of the peculiar shadows shown by the X-Ray. The physical signs were indeterminate, the heart signs and pressure symptoms slight but somewhat suggestive of aneurism and the autopsy showed small disseminated growths, bilateral in distribution though chiefly affecting the left lung.

upper sternum. These signs are usually so indefinite, occurring as they do in areas most unfavorable for percussion or auscultation, that the early diagnosis rests usually upon pressure symptoms and the knowledge of an adequate cause. (The subject of pressure symptoms is thoroughly discussed under the head of aneurism, page 205.) They are practically identical in all forms of mediastinal growth, though the most *distressing* cases ever observed by the author have been associated with the glandular enlargement of Hodgkin's disease.

Seek a cause.

MEDIASTINAL ABSCESS.—This excessively rare condition may be acute or chronic, and occurs chiefly in the male in connection with traumatism, the acute infectious diseases, pulmonary abscess, gangrene or advanced tuberculosis. It is recognized by the symptoms of sepsis associated with severe substernal pain and marked pressure symptoms. It may rupture and produce a fluctuating tumor in an intercostal space or discharge into the esophagus or trachea. The use of a *fine* aspirating needle usually makes the diagnosis.

Rare and obscure.

CHRONIC INTERSTITIAL PNEUMONIA (fibroid phthisis, pulmonary cirrhosis).—In spite of years of observation the conditions characterized by extensive fibroid changes remain imperfectly classified. In a broad sense it is best treated under the one heading and we may assume that lobar pneumonia, broncho-pneumonia, old pleurisies, syphilis, echinococcus cysts and tuberculosis may operate to produce a lesion, the chief characteristic of which is fibrosis. *Chronic diffuse interstitial pneumonia* is excessively rare as a sequence of acute lobar pneumonia though a few cases have been described with some accuracy. *Chronic broncho-pneumonia* is more common but is usually tuberculous. *An interstitial pneumonia* following pleurisy (pleurogenous) undoubtedly occurs as a result of prolonged compression, it being associated usually with a greatly thickened and adherent pleura. The cases of interstitial pneumonia due to continuous dusty occupations (pneumonokoniosis) and the syphilitic tuberculous and echinococcus forms are better understood.

Fibrosis.

Types

Morbid Anatomy.—The process may be lobar (massive) or lobular (peribronchial, broncho-pneumonic, insular). The sound lung is markedly emphysematous and the heart is thus pushed and drawn towards the diseased lung, pulmonary shrinkage and adhesions being usually marked, though sometimes lacking (e.g. in pneumonokoniosis). The lung itself may be marvelously shrunken and show chronic bronchitis, multiple bronchiectases and perhaps aneurismal dilatations of the pulmonary artery. The heart is enlarged, its right chambers being

Compensatory emphysema.

Retraction.

especially dilated. The varied nature of the lesion makes possible wide differences in post mortem findings. The most extreme cases represent the massive form of the disease, while gummata, apical tuberculous cavities or echinococcus cysts may indicate the specific primary cause as may the peculiar pigmentation of the tissue observed in cases of anthracosis (coal miner's disease) and siderosis (due to metallic particles), chalicosis (grinder's rot, stone cutter's phthisis).

Cardiac changes.

Anthracosis.

Siderosis.

Symptoms.—The symptoms accurately follow the morbid anatomy as stated, being those of emphysema, chronic bronchitis, bronchiectasis or chronic phthisis, according to the nature and extent of the lesions, combined with physical signs bearing the same relation to causation. The displacement of the heart, retraction and immobility of the affected side, scoliosis, and the depressed shoulder are emphasized by comparison with the voluminous opposite side. Symptoms of marked *infiltration* are usually found but vary widely with the degree of involvement. The disease is not only remarkably chronic but one which permits considerable activity for many years or even for a reasonably long lifetime.*

Usually marked.

PULMONARY SYPHILIS.—There are no characteristic symptoms of this disease which most commonly manifests itself as interstitial pneumonia. It occurs both in congenital and tertiary acquired syphilis. The so-called *white* pneumonia is of no clinical importance, being found only in the lungs of dead babes usually stillborn. The former process is frequently associated with tuberculosis. In acquired syphilis, gummata, single or multiple, varying in size from a small seed to a hen's egg may be encountered. The physical signs are in no way peculiar to syphilis. Whether there is an actual destructive disease of the lung and true syphilitic phthisis is still debatable. There are certainly rare cases in which softening is associated with caseous gummata and similar cases not infrequently occur in connection with tuberculosis.†

White pneumonia.

Tuberculosis and syphilis.

* One case observed by the author during a period of nearly 20 years has passed through several severe illnesses and has lived to see his two healthy brothers die of acute disease; another case, showing the classical signs of inherited syphilis and every evidence of massive chronic fibrosis, has undergone an appendicectomy and nephrotomy and several attacks of influenza. Both men have been almost continuously at work during the whole period.

† No one can have failed to encounter cases of undoubted acquired syphilis in which a rapidly advancing tuberculous process showed a marked improvement following the use of specific medication.

PULMONARY ACTINOMYCOSIS.—In all essential particulars this disease presents the picture of pulmonary tuberculosis and its diagnosis almost invariably depends upon the recovery of the specific organism from the sputum. It should be suspected if in such cases tubercle bacilli are absent, the bases chiefly or primarily involved and superficial swellings or brawny inflammations noted.

Differential factors.

ASPERGILLOMYCOSIS.—This rare disease is due to the aspergillus fumigatus and has been observed chiefly in those who handle infected flour, meal or grain. The symptoms are essentially those of pulmonary tuberculosis without the bacilli and the diagnosis can be made only by the discovery of the mycelium. It may also occur as a secondary disease in connection with the various chronic pulmonary ailments and may assume a predominatingly bronchial, nodular, or, cavernous form.

Resembles tuberculosis.

PULMONARY HYDATIDS.—Diagnosis.—So long as hydatid cysts are central and small, few or no symptoms are produced. If they enlarge, and particularly if they reach the pleura, cough and pain may be severe. Fever is usually absent, and dyspnœa slight. Physical signs may be baffling, indeterminate, or, those of consolidation or pleuritic effusion with the usual pressure displacements but weakened breath sounds and diminished fremitus. The area of dulness may be characteristically circular or with *signs* of *effusion* there may be marked *pleural friction* over the area of dulness as in Dr. Bristowe's case.* If rupture occurs the immediate symptoms may be urgent, the watery nature of the fluid is suggestive and characteristic and hooklets and fragments of the membrane may be present. Hydatid thrill and superficial rounded tumors may be found. Fowler emphasizes the *relative* absence of *mediastinal pressure* symptoms (see aneurism) despite the evidences of a *large tumor or exudate.*

Symptoms variable.

Peculiar cases.

Rupture.

CHYLOUS PLEURISY.—A chylothorax results usually from occlusion or rupture of the thoracic duct or receptaculum chyli, usually as a result of malignant or tuberculous disease. The diagnosis can only be made by the discovery of a pleural effusion and the removal of a portion for examination. *Such cases lack any history of acute pleurisy.*

Exploratory puncture.

MALIGNANT GROWTHS OF THE PLEURA.—What is said under the head of malignant growths of the lung applies here save that the primary signs and symptoms are *pleural.* Not only is pleural effusion simulated but in many instances actually present. Cardiac dis-

Effusion.

* Referred to by Fowler.

placement is usually less marked than in simple effusions but usually a differential diagnosis depends upon the withdrawal of blood stained fluid in amount disproportionate to, or without a proper diminution of, the area of flatness.

THE HEART AND BLOOD VESSELS.

THE ARTERIAL PULSE.—Whenever possible the pulse should be taken casually while talking of other matters and allowance made for the nervousness incident to examination and the effect of physical exertion. Caution.

Technique.—A correct technique is of the utmost importance and the patient's arms should be similarly placed in a position free from restraint, flexion, or muscular compression of the vessels. The pulse should be taken simultaneously in the two radials and three fingers applied lightly over the artery at the wrist.* Important factors.

Points to be Determined.—(1). *The size of the artery.* (2). *Pulse rate or frequency.* (3). *Regularity of rhythm.* (4). *Uniformity of strength.* (5). *Synchronism and equality of the right and left radial pulses.* (6). *The force required to obliterate them* (tension.) (7). *Abnormal thickening of the artery* (arterio-sclerosis).

When the physician's fingers are applied to the artery the first four points are determined almost unconsciously and instantaneously, the vessel being lightly rolled under the finger to get its size, and pressure made with the upper finger until the pulse is lost to the lower, the force exerted being the measure of *tension.* The empty artery is then rolled under the lower finger to detect any thickening of its walls (*arterio-sclerosis*) and *any vessel that can thus be felt as a distinct tube is sclerotic and hence abnormal.* Such may be merely palpable, distinctly rigid, or, carry tiny plaques of lime salts. Size and tension. Sclerosis.

Precautions.—It is ordinarily sufficient to count the pulse for 15 seconds and multiply by 4 to get the rate per minute, but if any abnormalities in rhythm, quality or force be present, it should be taken for at least one minute and if excessively rapid it may be necessary to count every second or third beat. Not infrequently, especially in certain valvular lesions, some systoles are inefficient, and, though readily auscultated, yield no pulse wave and in such cases nurses' pulse records are frequently worthless. *A recurrent pulse* is one that cannot be cut off by the pressure of the upper finger because of an unusually free commun- Special manœuvres. Vitiated records.

*There is no better proof of bad training than the gingerly one-fingered approach sometimes observed.

How detected.

ication and recurrent flow from the palmar arch. This may be obviated by making obliterating pressure with the lower finger and carrying out the usual procedure above, or, by compressing the ulnar artery. Faulty position of the patient's arm and hand may greatly modify any pulse.

Position of arm.

The forearm should be slightly flexed upon the elbow, the wrist thrown slightly backward, and very slightly supinated. A careless student frequently fails to note unequal force in succeeding beats or jumps at conclusions if he finds a unilateral weakening or absence of the pulse.

Weak pulse.

The former is most important evidence of deficient heart strength, the latter is ordinarily due to an abnormal course of the radial artery,

Misleading variations.

or more rarely, to actual blocking of the vessel or to aneurism, and the findings must always be checked by comparing the larger arteries, such as the brachials of the two sides. A *full bounding* pulse is frequently

A common error.

mistaken for a *high tension* pulse because of faulty technique. The correct determination requires much practice and for *extreme* accuracy demands the use of the special instruments.

Pulse Frequency.—Aside from mental excitement, the digestive process and bodily exertion, which latter includes even the slight disturbance due to readjustment of the position in bed or evacuation of the bladder or bowels, the position of the body affects the findings and in erect posture the rate is from 10–15 beats faster than when recumbent. The sitting posture shows a rate midway between the two.* The average

Exertion and attitude.

Age.

normal rates are for the first year of life from 130–140; from the first to the fourth year gradually dropping to 105 or 110; and so diminishing until at the fifteenth or sixteenth year it reaches from 75 to 80 beats per minute. During middle age and up to 60 years frequency is slightly

Sex.

diminished, sometimes increasing somewhat beyond that age. *Women show a rate from 5–8 beats a minute faster than men,* and it is slightly

Fever.

slower in tall than in short persons. *Fever.* Any abnormal increase in frequency suggests the use of the clinical thermometer and we find that as a rule *the rate increases from 8–10 beats per minute for each degree of temperature above the normal,* and further, that the behavior of pulse

Pulse temperature ratio.

in fever is of both diagnostic and prognostic importance.† In general an increase in the rate out of correspondence with the rule given suggests diminished resistance to the disease and constitutes a danger signal. In those few diseases in which the pulse rate should be lower than the

*This variation was formerly believed to be due to pulse tension as affected by posture, a theory exploded by the fact that the sphygmograph shows but a trifling variation in pressure.

†See Fever, p. 49.

rule, excessive rapidity has a still greater significance. *High temperature and relatively slow pulse are observed especially in tubercular meningitis, typhoid fever, yellow fever and lobar pneumonia,* as well as in febrile ailments associated with *some of the organic causes of slow heart, such as coronary sclerosis, myocarditis and aortic stenosis. In feverish children a relatively rapid rate is of far less significance than in the adult. A rapid pulse occurs in many non-febrile ailments,* most markedly in exophthalmic goitre and in the slight or marked incompensation stages of mitral stenosis and insufficiency, myocarditis and aortic insufficiency, and in acute endocarditis, pericarditis, and, the pressure displacements of the heart. In children an *excessively rapid pulse may suggest the onset of scarlatina* before the appearance of the rash and *in incipient pulmonary tuberculosis, an excitable and persistently over-rapid pulse is one of the most constant and significant of the early signs.* Aside from these organic disturbances must be considered sexual neurasthenia, the overuse of tobacco (often associated with palpitation), physical and mental overstrain and idiosyncrasy.

Diseases suggested.

Rapid pulse.

Tachycardia.—Mere excessive rapidity, paroxysmal or persistent, is known as tachycardia, and this term should be strictly limited to the pulse free from irregularity, intermittency or abnormal accentuation of the heart sounds. An excessively rapid "*running*" pulse associated with marked evidence of cardiac weakness and suggestive lack of accentuation is a forerunner of death in certain diseases.

Bradycardia (brachycardia).—A very slow pulse may be physiologic but most of those thus described have subsequently been proven to be due to organic disease of the heart or brain. Excessively low count (20 beats to the minute) *may* be associated with coronary sclerosis, fatty infiltration and injuries. Lesser degrees of slowing may be met with in aortic stenosis, chronic myocarditis, cerebral hemorrhage or tumor, meningitis, epilepsy, general paralysis of the insane, melancholia, mania, myxœdema, jaundice, uræmia, exhausting disease and sometimes, acute ailments, especially diphtheria, in which no doubt myocarditis is really chiefly accountable. In the so-called *Stokes-Adams syndrome* a bradycardia is associated with epileptiform, syncopal, or apoplectiform attacks. As regards its occurrence in acute infections, during either the active stage or convalescence, the rule may be laid down that *any sudden and decided drop below the normal or usual rate unless associated with crisis is quite as much, if not more suggestive of danger and of need of special watchfulness and care as would be excessive rapidity.*

Usually indicates disease.

Important inference.

Scope of terms.

Irregularity and Intermittency.—*Irregularity* includes variation in volume and strength as well as wave intervals, though the former are better described by the term unequal; by *intermittency* is meant the omission of beats and the two conditions are often confounded by nurses and students. The stethoscope shows that intermittency may be due either to inefficient or absent systole. Both conditions may be temporary and trivial and due to purely functional disturbance or the temporary action of such substances as tobacco, tea and coffee. In the absence of definite clinical findings their significance is far more serious in middle aged, or elderly people than in the young and both variations admit of subdivisions. *Irregularity is frequently combined with inequality and excessive frequency* (delirium cordis) *and may be transient, variable, persistent or periodic.* A strong beat may alternate with a feeble one (*pulsus alternans*) or double or triple beats may occur (*pulsus bigeminus, pulsus trigeminus*), or, the normal variation due to deep inspiration may be so exaggerated as to make the radial pulse weak or imperceptible during inspiration (*pulsus paradoxus*).

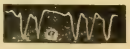


Fig. 54. Marked irregularity. Low tension.

Significance.

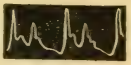


Fig. 55. Bigeminal pulse.

Varieties.

Inequality.

Idiosyncrasy *is a possible factor and the greatest stress should be laid upon the age of the individual and the persistence of arrhythmia as compared with transient disturbance.*

Inference from Arrhythmia.—The following *possible* conditions must be considered in every case. (1). *Failure of compensation in heart lesions* whether due to arterio-sclerosis, myocarditis, fatty degeneration or overgrowth, acute dilatation or valvular disease. (2). *Certain stages of intracranial disease or disturbances* such as mental overwork and strain, cerebral meningitis, tumor, abscess, softening and hemorrhage or actual cranial injury. (3). *Sexual excess and the overuse of alcohol, tea, coffee and tobacco.* (4). *The toxines of diseases* such as pneumonia, scarlatina, diphtheria and typhoid. (5). *The over-action of drugs* such as aconite, belladonna, digitalis, cocaine and morphine. (6). *Renal disease, exophthalmic goitre,* the terminal stages of *exhausting ailments* or *early stages of tuberculosis* in which however rapidity and an easily excitable heart are chiefly observed. (7). *Digestive disturbances* especially those attended by flatulence.

Important data.

The *paradoxic pulse* is most frequently observed in excessive cardiac weakness, especially if associated with obstructive pressure such as may be exerted by pericardial effusion, mediastinal tumor (whether aneuris-

mal, glandular or malignant), indurative mediastinitis or laryngeal, tracheal or bronchial stenosis.*

Pulsus Celer and Pulsus Tardus.—The finger, and better still the sphygmograph differentiates a wave or quick rise and descent (pulsus celer) from that of slow rise and fall. The former is exemplified by the water hammer pulse of aortic insufficiency, the latter (pulsus tardus) by the high tension pulse of arterio-sclerosis, interstitial nephritis, angina pectoris or aortic stenosis. Exact equality in the time of rise and fall is sometimes noted in thoracic aneurism and Sahli rightly affirms that even the finger may distinguish between the pulse that is celer in its ascent and tardus in its descent as in lesser grades of arterio-sclerosis.

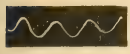


Fig. 56. Aneurism.

Disease associations.

Full Pulse.—The artery is full *between beats* and may be large or small and of high or low tension.

Large Pulse.—The *excursion* of the artery *is marked* as seen in aortic regurgitation (pulsus magnus et celer). It may be full as in fever (*bounding pulse*) but is usually of low tension. *Pulsus vacuus.* This is typified by the unsustained quality of the water hammer pulse, less so in mitral stenosis and extreme malnutrition, severe anæmias and neurasthenia. *The Small Pulse.* Aortic and mitral stenosis, myocarditis and aneurism furnish the best examples. *The thready or running pulse.* This small empty rapid pulse is a forerunner of death in most instances. *Wiry pulse.* This *small hard* pulse is best marked in peritonitis.

Unilaterally Weakened or Absent Pulse.—Unilateral retardation, weakness, or extinction, points to pressure, aneurism, thrombosis, embolism, wounds, or abnormal arterial distribution. *In aneurism of the ascending portion* of the arch involving the innominate, the right radial and carotid are affected, in *aneurism of the descending aorta* the left radial; or, a delayed pulse may accompany aneurism of the transverse portion.† *Too much reliance should not be placed upon this sign of aneurism as it is often lacking or without exact significance.* Loss of the pulse at the ankle may be associated with intermittent claudication or be a precursor of senile gangrene.

Important inferences.

Dicrotic Pulse.—A second beat or echo may be felt more or less

*The essential primary inference in any case is a weak peripheral circulation.

† Direct sac pressure, abnormally constricted or deformed openings, or, the complete or partial conversion of intermittent into continuous flow by the aneurismal sac make many variations possible.

Exhaustion.

distinctly under proper pressure and indicates ordinarily extreme exhaustion or toxæmia, early noted in typhoid fever and the typhoid state, but often found in exhausting diseases of various sorts, and, rarely, in health. **The Capillary Pulse.**—*A large pulsus celer produces capillary pulsation.* It is a rhythmic blushing and paling which may be *observed* over inflammatory areas, in the matrix on making light pressure over the nails or in the forehead or chest, ears, etc., by reddening the skin by friction. By steadily pressing a glass slide or even the rim of a goblet upon the mucous membrane of the lips its rhythmically oscillating border is readily seen and it may even be observed in the retinal arteries. *It is a valuable sign of aortic insufficiency but may also be observed in exophthalmic goitre, neurasthenia with anæmia, certain cases of fever, chlorosis, pernicious anæmia and acute hemorrhages.*

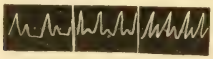


Fig. 57.—Dicrotism.

Important and suggestive.

DETERMINATION OF BLOOD PRESSURE.—Fortunately for the clinician blood pressure may now be measured quickly and exactly by simple and relatively inexpensive instruments nearly all of which depend upon the same general principles, i.e. the translation of the pressure required to obliterate the arterial pulse into the height of a mercury column in a manometer tube (see fig. 58). The portable instrument of Janeway* is accurate and practical because of its broad arm piece which excludes the errors (high readings) inherent in the narrow compression bands when applied to unusually fat or muscular arms, and because of its accurate regulation of pressure by a stopcock.

Simple devices.

Janeway's instrument.

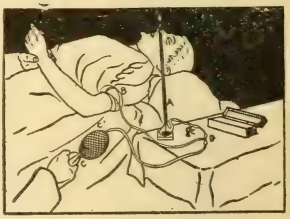


Fig. 58.—Cook's modification of the Riva-Rocci sphygmomanometer.

Technique.—The hollow armlet applied midway between shoulder and elbow is inflated by the hand bulb until the radial pulse is lost, then by the outlet thumb screw the pressure is lowered until the pulse return is just perceptible. As the pressure is equal in all parts of the closed system the height of the mercury column in the manometer tube is an exact index and the reading represents the "*maximum*" or "*systolic*" pressure. "*Diastolic*" or "minimum" pressure is determined by noting for ten or

Easy of application.

Terms employed.

*Note.—The reader is referred to Theodore C. Janeway's admirable monograph for an extended discussion of this topic. "The Clinical Study of Blood Pressure."

twelve pulsations the increasing amplitude of the pulse wave registered by the mercury column as the pressure is reduced in 5 mm. series. The point causing maximum excursion is the *index of diastolic pressure.* Below that is a limited pressure area of equal amplitudes. The "*mean*" pressure represents the average of systolic and diastolic readings. Diastolic readings run about 25–40 below systolic and in low tension vary 50–80 and in aortic regurgitation up to 100 mm. A loose band or a rapid or excessively small pulse makes diastolic pressure determination impossible and in every case the arm band should be closely adjusted, the arm supported at the heart level and the same position taken for a series of tests. *The limit of error in calcareous arteries is but 5–10 mm. and is negligible or easily estimated. The same figures represent the difference between females and <males and the standing> and sitting posture.* In *normal pressure* four factors are concerned, viz.:—*the initial heart energy, peripheral resistance, blood volume and the elasticity of the vessels.* The normal readings according to Janeway are:—for young adults. 100–130; older adults, 100–145; children, 90–110; infants under 2 years, 75–90. Excitement may cause a rise of 40 mm. and concentrated physical effort a slight increase.

Variations.

Sources of error.

Sclerosis.

Sex and attitude.

Factors involved.

Normal readings.

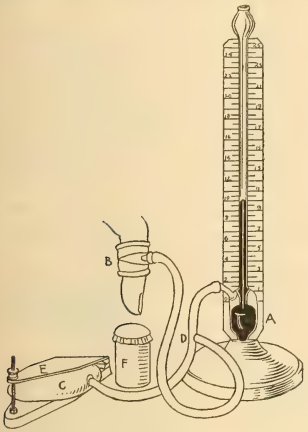


Fig. 59.—Gartner's tonometer.

Abnormally High Pressure.—In *chronic interstitial nephritis* the elements of arterio-sclerosis, high peripheral resistance and increased heart energy bring about high readings. In abnormal nervous excitement temporary high pressure may be observed. In secondary myocarditis *with associated arterio-sclerosis*, the uræmic state, lead poisoning, gout, cerebral hemorrhage and sometimes in *parenchymatous nephritis or early acute nephritis* high readings are present.

Various factors.

Abnormally Low Pressure.—The conditions giving the lowest readings are *shock, collapse and concealed hemorrhage.* In *visible* hemorrhage attended by nervous excitement, fear, and apprehension the pressure is raised. *The acute infectious diseases, anæmias and cachexias and the terminal stages of all diseases show low pressure.*

The Sphygmograph and Sphygmogram.—The sphygmograph has

undoubted value in connection with pulse taking, yielding in certain directions more important information than may be derived from touch and making if desired a permanent record. But although its use does not involve *much* time, the relation of time lost to *information gained* makes it unlikely that it will ever be extensively used in clinical work outside of hospital or consultation practice. *Certainly it cannot replace ordinary pulse taking*, if for no other reason than its failure to show the size of a pulse, and the condition of the arterial walls. The forms of sphygmograph in most common use are those of Marey and Dudgeon, either of which gives excellent tracings. A detailed description is unnecessary, the principle of all sphygmographs being that of the transmission of the pulse wave to a lever so arranged as to register the excursion of the compression pad upon some substance such as smoked paper which is passed under the point by clock mechanism at a known rate per second. *The difficulties attending the operation of these instruments and the effect of the personal equation in interpretation have been unduly magnified;* a little practice suffices and the differences in results are not so great as to obscure their clinical value. *To make permanent records* special paper may be obtained of a smooth, highly calendared surface which is easily smoked (not scorched) over the flame of burning camphor or even an ordinary candle. This, after tracing the name of the patient and the date, is dipped into a solution of photographer's varnish or similar substance and dried. The instrument may be used with or without the wrist band but the hand must always be supported *at heart level.* Before starting the clock work the pad should be, *and remain,* exactly over the artery and the milled head turned until the needle shows its maximum amplitude of movement;* the hand itself, slightly dorsiflexed, should rest steadily palm upward, with the fingers slightly bent.

Useful.

Not indispensable.

Principle.

Easy of application.

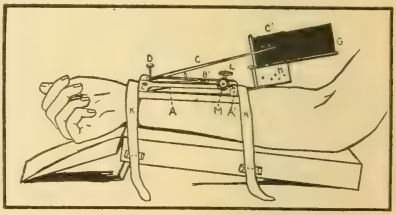


Fig. 60.—Marey's sphygmograph.

Caution.

Sphygmogram.—*The analysis of all tracings depends upon a thorough understanding of that of the normal pulse.* In this we recognize an *anacrotic or ascending limb* which at its summit or apex becomes the

Elements.

* It is usually best to make several tracings under slightly varying pressures.

katacrotic or descending limb, the latter being divided into two chief waves, the higher being the *tidal or predicrotic*, the lower the *recoil or dicrotic* wave; between these two elevations on the descending limb is a hollow representing the aortic closure; *the apex height shows the maximum excursion, the two secondary elevations* on the descending limb *the reactive contractions* of the artery.

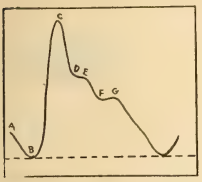


Fig. 61.
Normal sphygmogram.
b.c. percussion upstroke.
b. c. d. percussion wave.
d. e. f. tidal wave. g. dicrotic wave.

Normal tracings show a nearly vertical upstroke of moderate amplitude, a gradual descent and a moderately sharp apex, the tidal wave being small, the dicrotic low but well marked.

Abnormal tracings show:—(a). *Broad apex.* This indicates high tension and a strongly acting heart, aortic stenosis, aneurism, or too great pressure of the pad. (b). An *unduly long sharp apex* indicates low tension, and is especially marked in aortic regurgitation. (c). A *short upstroke* indicates small volume; as in mitral regurgitation, aneurism, aortic stenosis, and arterio-sclerosis. (d). A *long upstroke* means free peripheral circulation, sharp systole, or in general, low tension. Aortic regurgitation furnishes a typical example. (e). *Excessive obliquity* of the upstrokes points to slow filling of the artery and is especially noticed in mitral stenosis or regurgitation, arterio-sclerosis, aneurism or a weakened myocardium. (f). A *vertical upstroke* indicates large blood volumes and quick systole whether strong or weak and is exemplified by aortic regurgitation. (g). A *marked tidal wave* suggests high tension or actual obstruction as in aortic stenosis orarterio-sclerosis with a strong cardiac impulse. (h). A *diminished tidal wave* indicates cardiac weakness, or, *strength* with relaxed peripheral circulation, as in mitral or aortic regurgitation. (i). *Marked dicrotic wave* points to low tension or to high tension with cardiac weakness. It is best marked in the pulse of typhoid fever. (j). *Lessened dicrotic wave.* Aside

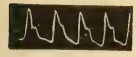


Fig. 62.—Aortic regurgitation.

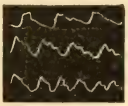


Fig. 63.—High tension.

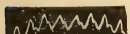


Fig. 64.—Undulating base line.

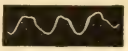


Fig. 65.—Arterio-sclerosis.

Fig. 66.—Irregularity.

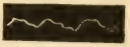


Fig. 67.—Aneurism.

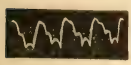


Fig. 68.—Pulsus biferiens.

Fig. 69.—Aortic stenosis.

from aortic regurgitation this points to an obstructed circulation with a strong systole, i.e. aneurism, arterio-sclerosis and aortic stenosis. Aside from these points, *irregularities in the line of descent* are noted in mitral lesions; *irregularities of the base line* corresponding to respiration in dyspnœa or any form of irregular forcible breathing; and any *intermittency, irregularity or hemisystole* is made clearly evident by the tracings. The plates accompanying this section clearly show the characteristics mentioned in the foregoing paragraph and it will be seen that an observer who thoroughly understands the principles involved can obtain information of considerable diagnostic, prognostic and therapeutic value.

Fig. 70.—Arterio-sclerosis.

VENOUS PULSE.—When distinguishing between the physiologic *negative pulse* and a pathologic *positive pulse* the external jugular is the vessel usually examined as being most superficial and accessible.

Undulant pulse.

The negative pulse disappears above and diminishes below the finger compressing the external *and internal* jugular veins, or, better still, both these and the subclavian, and is apparently due, not to direct

Systolic collapse.

regurgitation of the blood, but the transmission of a suction wave from the heart to the vein. Neither is it dependent upon destruction of the venous valves as its direction coincides with the normal blood flow through them, nor transmitted from the artery, else it would persist peripherally. Coincident sphygmographic tracing of the carotid pulse and the negative venous pulse proves the latter to be diastolic.

Positive systolic pulse.

By the same method *the systolic venous pulse*, proof positive of tricuspid regurgitation, is shown to be a *systolic regurgitant pulse.* It is chiefly apparent in the bulb, though occasionally peripheral and in extreme

Hepatic pulsation.

cases, especially in children, there is *coincident expansile pulsation of the liver* as a whole, most easily recognized by placing one hand upon the right lower thoracic zone, posteriorly, and the other over the enlarged liver in front. This manœuvre is necessary to eliminate the spurious transmitted pulsation frequently observed in cardiac dilatation.* *The positive venous pulse is lost peripherally but unaffected or increased centrally by compression.*

Diastolic Venous Collapse.—This rare sign indicates extensive pericardial adhesion and is due to diastolic recession of the chest wall accompanied by diastolic intra-thoracic suction. In appearance it

Resembles positive pulse.

resembles the positive (systolic) venous pulse, but under obliterating

*Sahli reports a case in which localized inflammatory pulsation of the liver occurred as the result of a suppurative cholangitis.

pressure the wave is *centrally* diminished or lost.* Compression should include both internal and external jugular veins in all attempts to differentiate the venous pulsations.

Positive Penetrating Venous Pulse.—Very rarely and usually in association with a capillary pulse a pulsation may be observed in the terminal venous radicles. Compression causes central obliteration but does not affect the peripheral portion.

Other Visible Respiratory Venous Phenomena.—The swelling of the veins of the neck during violent straining or sustaining a musical note is familiar to everyone and is seen in all forms of severe dyspnœa, whether temporary or permanent. In chronic conditions the overdistension becomes permanent and occasionally the usual *inspiratory diminution and expiratory fullness* is reversed, in which case indurative mediastinitis, chronic pericarditis, pleuritis, aneurism or any other form of mediastinal tumor is suggested. Students often find difficulty in distinguishing between *venous pulsation* of the internal jugular *and carotid pulsation; the former covers a wider area and is undulatory and deliberate in character, and pressure upon the carotid does not affect it.*

Common form.

Rare form.

EXAMINATION OF THE HEART AND GREAT VESSELS.

Inspection.—This should be both direct and tangential, general and local. The most important regions are: (a). *The valvular areas* described on page 84. (b). The *manubrium and its neighborhood.* (c). The *area lying between the inner edge of the left scapula and the spinal column.* One seeks chiefly to note:—(a). *Abnormal pulsations.* (b). *Asymmetry.* (c). *Tumors.*

The Apex Beat.—This should be represented by a gentle rhythmic uplifting of the fifth interspace in an area not more than an inch in diameter, well within the mid-clavicular line. It is not always present in health, either because of overlying fat or an intervening rib and the beat is often excessive even in the case of a normal heart, because of temporary excitement, exertion, indigestion, narcotism, neurasthenia, etc. A heart that is abnormally strong by reason of hypertrophy causes a distinct and wide spread *heaving* (uplifting) impulse, and *the beat of a badly dilated heart is wavy, diffuse and indeterminate.* In either case the area of pulsation is greatly increased. **The Position of the Apex Beat.**—Abnormalities of position must inevitably

Area and position.

Force.

Heaving vs. Wavy.

*The author has inserted this statement because of the weight of authoritative opinion but believes it to be subject to exception.

follow decided changes in the cardiac outline or actual displacement of the heart.

Important in diagnosis.

Displacement of the Apex Beat.—*Upward:* (a). High position of diaphragm. (b). Tympanites. (c). Ascites. (d). Abdominal growths. (e). Pericarditis with effusion (often at nipple level). *Upward and to left:*—Effusion into right pleural sac. (a). Liquid. (b). Gaseous. *Downward:*—(a). Aortic aneurism. (b). Mediastinal tumor. (c). Senility. (d). Hypertrophy of left ventricle. (e). Collapse of abdominal viscera. *To right or left:*—(a). Hypertrophy and dilatation. (b). Effusion of gas or liquid into pleural sac—i.e. pneumothorax, hydrothorax, etc. (c). Unilateral emphysema. (d). Pleural adhesion and retracted lung. (e). Marked solid enlargements of lung or of the left lobe of the liver.*

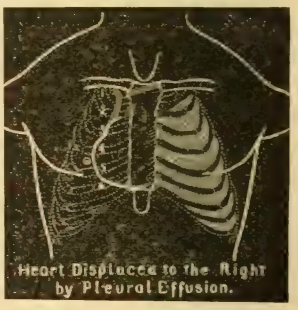


Fig. 71.

Often misinterpreted.

The apex beat may be invisible *by reason of:*—(a). Interposition of rib. (b). Fat chest wall. (c). Feeble heart. (d). Emphysema. (e). Edema of chest wall. (f). Pericardial effusion (marked). (g). Pleural effusion. (h). Transposition of the viscera (is present on right side). **Its area and apparent force may be increased** *because of:*—(a). Nervous excitement. (b). Hypertrophy or dilatation. (c). Retraction of lung.

True or false.

Systolic Retraction.—This may result from dilated heart, old pleuro-pericardial adhesion, or, shrinkage and contraction of the left lung. It is one of the signs of adhesion of the pericardium to the heart itself, which causes a systolic drag upon the diaphragm but to be of diagnostic value in this connection it should involve the posterior interspaces as well.

Misleading.

Other Precordial Pulsations.—A strongly acting nervous heart and thin chest wall may cause systolic pulsation in the third, fourth or fifth left interspace and in the upper portion of the epigastrium even though the heart be normal, but ordinarily it is associated with cardiac dilatation, adjacent pulmonary cavity, pulmonary fibrosis, emphysema of the lungs, anæmia, neurasthenia or excessive emaciation.

Pulsation in the Region of the Manubrium.—Such an impulse

* *Respiratory* lateral displacement may occasionally be *evident* in right sided pleural effusion. See p. 134.

if marked always *suggests* aneurism of the aortic arch, and pulsation of the manubrium itself is of special significance, because of its association both with aneurism and certain forms of mediastinal growth. Mediastinal tumor.

Precordial Bulging.—Aside from the presence of actual growths this is usually due to one of two causes, viz., aortic aneurism, or, excessive enlargement of the heart. It is most marked in those cases originating in early childhood in which the growing osseous structures have yielded readily to the pressure beneath. One may then often fix the date of the heart lesion seen in advanced life, and find perhaps, that it has existed for a half century. Value in diagnosis.

Palpation.—Palpation serves to confirm inspection and to detect *thrills* associated with valvular lesions or anæmia, *points of tenderness* and the general characteristics of tumors of any kind. It is of special Scope and value.

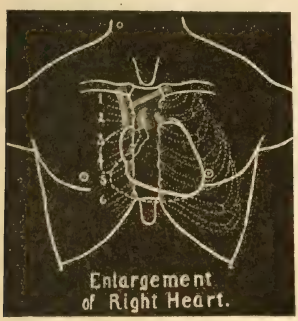


Fig. 72.—Dotted lines indicate percussion area. (Auscultatory.)

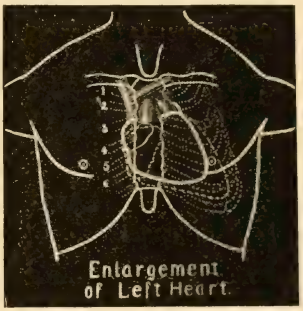


Fig. 73.—Dotted lines indicate percussion area. (Auscultatory.)

value in connection with *expansile pulsation and diastolic shock* of aneurism and the *presystolic thrill and systolic shock* of mitral stenosis. No examination of the diseased heart is complete that does not include the palpation of such related organs as the lungs, liver and spleen and determine the question of meteorism. Caution.

Percussion.—*Percussion is often decisive in the differential diagnosis of heart lesions.* One should always percuss from the more resonant to the less resonant areas, save in determining the superficial dulness, and seek to determine (a) *the deep or relative cardiac dulness*, (b) *its superficial dulness*, (c) *the resonance of the region of the aortic arch.* Abdominal distension and pulmonary, hepatic, or splenic engorgement are also important factors if the heart is abnormal. If ordinary percussion be used the anatomical border of the heart is not accurately Never omitted. Cardiac boundaries.

outlined, but by auscultatory percussion, this may be very closely approximated (see percussion and auscultatory percussion, pp. 92, 98), and the student should accustom himself to the sound of the normal dull tympany of the manubrium sterni, as this is one of the most important regions. In determining the superficial cardiac area it is best to commence at its known centre and percuss from that in radiating lines.

Manubrium.

Significant Changes in Percussion Area.—An increase in the area of superficial dulness may mean (a). *Enlargement of the heart.* (b). *Retraction of the lung.* (c). *Pericardial effusion.* Changes in the shape of the relative cardiac dulness usually indicate enlargement of the heart itself, suggest the individual chambers involved and hence the underlying valvular lesions (see plates 72, 73, 74). As will be seen later, *such changed outlines serve as a check upon the diagnosis of valvular lesions and pericardial effusions, and the student must never forget that serious dilatation thus indicated may be unattended by any valvular murmurs.* He should also carefully note and localize any pain or tenderness attending this procedure. Syphilitic periostitis, for example, may be encountered over the sternum and ribs and might prove of the utmost importance in a diagnosis.

Of utmost value.

Murmur may be absent.

Tenderness.

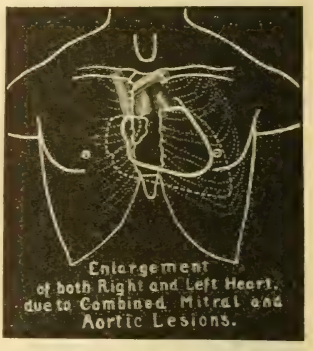


Fig. 74.—Dotted lines indicate percussion area. (Auscultatory.)

Auscultation.—This determines (a). *The condition of the heart muscles.* (b). *The competence and condition of its valves.* (c). *The tension present in the aortic and pulmonary circuits.* (d). *The condition of the lungs (congestion, edema, effusions).* (e). *Abnormalities of the aortic arch.* (f). *The presence or absence of murmurs in the tributary veins and arteries.* The first and most important consideration is the *quality of the heart sounds* in the valvular areas and in the carotid arteries. As in the case of the lung, a common mistake in the teaching of students lies in the failure to drill them thoroughly in the normal characteristics of, or common variations in, the heart sounds, and *students and physicians alike are too often satisfied if no murmur is heard* and do not demand normal heart sounds and accentuation. *In no other way can one explain the frequent failures to recognize the serious meaning of abnormal quality and accentuation, decided diminution or*

Wide diagnostic scope.

Know the normal.

Avoid blunders.

entire absence of heart sounds, conditions associated with some of the most serious organic lesions of the heart.

HEART SOUNDS.—The First Sound.—A thorough understanding of the mode of production and normal accentuation of the heart sounds is absolutely essential to the intelligent interpretation of heart murmurs. *At the apex the normal accentuation is on the first sound; at the base on the second.* Disregarding conflicting theories and fine distinctions, one may say that *the essential elements in the production of the first sound are the contraction of the ventricular walls and the coincident closure of the auriculo-ventricular valves.* It follows that *abnormally increased accentuation of the first sound, as heard in any of the four auscultation areas, indicates an overacting ventricle* whether due to mere increase in strength, as in hypertrophy from valvular disease, nervous overaction, excessive contraction of a partly filled ventricle, as is the case in mitral stenosis, or to heightened arterial tension, as in arterio-sclerosis, chronic toxæmia and interstitial nephritis.

Normal accentuation.

Production of sounds.

Abnormal accentuation.

The Second Sound.—*The second sound is produced by the simultaneous closure and tension of the aortic and pulmonary valves immediately following ventricular contraction.* Hence this second sound must primarily depend upon the integrity of the valves themselves, and measure the amount of that recoil which is the resultant of the forces of propulsion and the resistance encountered in the artery. The aortic valve being sound, any *increase in general arterial tension will produce an accentuation of the aortic second sound,* and *similar increased tension in, or obstruction to, the pulmonary circulation will result in an accentuation of the pulmonary second sound. The degree will depend largely upon the soundness of the valves, the strength of the ventricles and the integrity of the mitral and tricuspid leaflets.**

Cause.

Accentuation.

Practical Application.—Much information is afforded in both acute and chronic disease by a study of the variations in accentuation. For example, lobar pneumonia must inevitably be accompanied by marked accentuation of the pulmonary second sound and a dangerous tendency to dilatation of the right ventricle. If this dilatation becomes extreme the pulmonary second sound is markedly weakened and if tricuspid regurgitation occur it may be wholly lost, a sign of the gravest import. The pulmonary congestion incident to mitral stenosis and regurgitation makes accentuation of the pulmonary second sound an important diagnostic feature and as in pneumonia, a loss of this accentuation may

Diminished 2d sound.

Accentuated 2d sound.

*The mitral and tricuspid tones are increased under the same conditions but to a less marked extent.

Modified 1st sound and aortic 2d.

be a serious symptom. *The first sound, as heard at the apex, may be wholly lost or replaced by a murmur in mitral regurgitation, and both aortic stenosis and incompetence tend to obscure the second sound in the aortic area and in the carotid artery. In mitral stenosis the aortic second sound is weakened,* the mitral being accentuated and sharp or slamming unless marked regurgitation coexist; in aortic regurgitation the first sound at the apex is obscured *and in combined mitral and tricuspid regurgitation* all sounds are greatly diminished. Mere weakening of both sounds, especially at the apex may be due to a fat or edematous chest wall, emphysema or pericarditis, or, may suggest cardiac displacement. In fibroid phthisis, pleural effusion, mediastinal growths, diaphragmatic hernia or even extreme meteorism or ascites, the cardiac displacement may be suggested by the changed site of the audibility of the sound or murmurs. *Furthermore one can seldom with certainty assign murmurs heard under such conditions to the individual valves—as they may wholly or in part disappear when the dislocation is corrected.* Pleuritic effusion offers the most frequent examples.

All sounds faint.

Displaced heart.

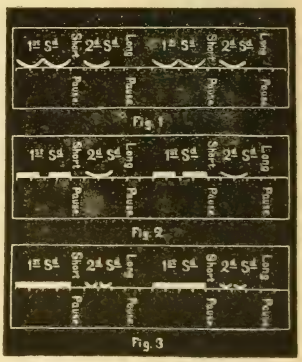


Fig. 75.—Division and Reduplication of the Heart-sounds. Fig. 1. Reduplication of the first sound. Fig. 2. Division of the first sound. Fig. 3. Division of the second sound.

Increased Audibility.—Aside from causes already given this may result if, from any cause, the lung is retracted from the heart, and hence at times coincides with a change in the site of *maximum audibility*, e.g. fibroid lung or adjacent excavation. Sounds are occasionally transmitted loudly and over wide areas by pulmonary consolidation or cavity, and, in pneumo-pericardium.

Changes in Timbre.—A tone may be *harsh, ringing, "slamming," "fœtal" or metallic.* Metallic clacking is frequently audible in palpitation, especially if the stomach be distended by gas. *Persistence* of such qualities usually means disease of the valves or heart muscle, or, high arterial tension. Fœtal sounds and a slamming first mitral are always pathologic.

Important.

Reduplication of Heart Sounds.—*The normal heart sounds consist of two systolic and two diastolic sounds so blended as to form one systolic and one diastolic tone because of the synchronous closure of the valves*

of the right and left heart. Under many conditions this synchronism is so interfered with as to produce doubling or actual division of one or both sounds. The phenomenon of reduplication has aptly been compared to the sound produced by the closure of double swinging doors. A normal heart, if temporarily overacting, may be the seat of doubling or splitting of the heart sounds; but in general it may be said that hearing a reduplicated *second sound* or triple rhythm at the apex, one should suspect the presence of mitral stenosis* or myocardial degeneration. *Gallop rhythm* is a form of reduplication *heard over the whole heart* though sometimes more plainly over one side (Potain) that may be graphically represented. It is usually strongly suggestive of marked incompensation, interstitial nephritis, emphysema, arterio-sclerosis and exophthalmic goitre. *Fœtal or pendulum rhythm.* In this the heart sounds are rapid and equally distant, i.e. if as is usually the case there is lack of the characteristic resonance, they resemble the actual fœtal heart sounds and the condition has been well named "embryo-cardia" (Stokes–Von Huchard). The expert clinician recognizes various modifications and minor degrees of the utmost significance and importance *as indicating a marked enfeeblement of the heart muscle.*

Occasionally trivial.

Usually pathologic.

Inferences.

A serious sign.

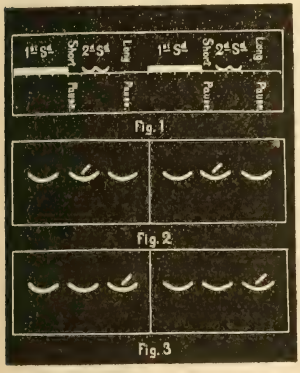


Fig. 76.—Reduplication and Division of the Heart-sounds. Fig. 1. Reduplication of the second sound. Figs. 2 and 3. Gallop rhythm.—(*After Vierordt.*)

Auscultation Areas.—The four surface areas for auscultation are:—(1). *The mitral area* (apex). (2). *Tricuspid area* (lower half of sternum). (3). *Pulmonary area* (second left intercostal space). (4). *Aortic area* (second right intercostal space). These points do not correspond to the exact location of the valves, which may all be included in the area of a large stethoscope bell, but represent the areas in which the sounds of the respective valves or their associated murmurs are best heard.

Clinical areas.

Heart Murmurs.—A heart murmur is an extraneous sound of cardiac site tending to obscure or partly or wholly replace the normal heart sound and directly related to it in rhythm.

Definition

*Less commonly in regurgitation unless associated with myocarditis of marked degree or arterio-sclerosis.

Common Sources of Error.—It should never be forgotten that the sounds produced by an overacting, tumultuously beating heart can seldom be accurately interpreted. Under such conditions, existing organic murmurs may be obscured or be unplaced in rhythm; or, as more commonly happens, murmurs may be present, purely temporary in character, which may be and often are regarded and treated as organic. Such *transient dynamic or accidental murmurs and harsh heart sounds associated with abnormal accentuation are extremely common.* They call for rest, reassurance, the administration of such drugs as digitalis and the bromides, and *always* for repeated examinations. *All patients should be examined if possible both when recumbent and when erect,* as the murmurs of regurgitation are usually best heard in the former position, those of stenosis in the latter, while sometimes, as in the case of mitral stenosis, a murmur may only appear at the moment when the patient reaches the upright position and then rapidly subside. The diastolic murmur of aortic regurgitation may be increased by raising the arms above the head, or, require brisk exercise to make it audible.

Transient murmurs.

Attitude important.

Useful manœuvres.

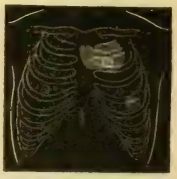


Fig. 77.—Anæmic Murmurs.—(*Sansom.*) Coexisting pulmonary and apex murmurs. (Nine per cent. of cases.)

Fig. 78.—Anæmia.—(*Sansom.*) Murmur over right ventricle and conus. (Eleven per cent. of cases.)

The Intensity of the Murmur.—The stronger the heart and the narrower the opening, the louder is the murmur, and *speaking broadly* it may be said, that *the louder the murmur the better is the prognosis.* Cases yielding soft murmurs elicitated with difficulty, or those in which no murmur is present though the heart is incompetent, are usually of the most serious import. The rule is of course subject to many exceptions.

Loud vs. Soft.

Anæmic or Hæmic Murmurs.—*The murmurs associated with anæmia are systolic in time and soft and blowing in character,* are seldom widely transmitted. and are usually best heard over the pulmonary area* (second left intercostal space). They are heard less frequently over the apex, aortic area, etc., and differ from the true

Time, quality and site.

* Rarely they may be loud and harsh as in a case of exsanguination due to acute hemorrhagic gastric ulcer observed by the author. In this case the murmurs were auto-audible and to be heard with the ear several inches from the chest.

murmur of mitral regurgitation in the fact that they have not the same transmission to the axilla and back. So also when heard over the aortic area (second right intercostal space), they lack the transmission into the carotids and subclavian characteristic of true aortic stenosis. Their association with anæmia and disappearance under appropriate treatment further serve to differentiate them in practice. Associated dilatation is uncommon, and when present, is slight. A few cases of diastolic hæmic murmur have been reported.*

Lack transmission.

Associated anæmia.

Therapeutic test.

Fig. 79.—Anæmia.—(*Sansom.*) Murmur in aortic area. (Eleven per cent. of cases.)

Pleuro-pericardial Murmurs.—When the apposed layers of the pleura and pericardium become inflamed in the extension of a pleuritis or pericarditis, a murmur may be present which is usually distinctly frictional in character, and heard best in inspiration. This sometimes endures for a long time after the original disease, usually lacks the quality and transmission of a valvular murmur, but often closely simulates pericardial friction *per se*. By holding the breath in full inspiration the inflamed surfaces may separate and the murmur be lost *if the area involved* is sufficiently small. So also the differences noted in deep breathing may be much greater than pericarditis alone could explain.

Manœuvres.

Fig. 80.—Anæmia.—(*Sansom.*) Usual site of murmur—pulmonary area. (Fifty-nine per cent. of cases.)

Other Forms of Accidental Murmurs.—A *cardio-respiratory murmur* is a systolic *whiffing* murmur heard best during inspiration and in the region of the heart border. Another cardio-respiratory murmur not a friction murmur is that heard in full *expiration*, in the same area, and despite authoritative opinion to the contrary, the author believes these to be often of great significance and, importance, and has again and again found them in association with the lesions of pulmonary tuberculosis, both in its ancient and arrested, and, in its incipient and active form. He does not pretend to deny, however, that such a murmur often has little significance. Some curious *crackling murmurs* may be

Many suggest present or past disease.

Fig. 81.—Anæmia.—(*Sansom.*) Systolic apex murmur. (Seven per cent. of cases.)

*One reported by the author occurred in the terminal stage of pernicious anæmia.

Curious crackles.

heard along the left edge of the sternum, particularly, in connection with advanced emphysema. Similar sounds are heard over the right ventricle at a point between the apex and the sternum to which various unsatisfactory explanations have been assigned. If the pericardium

Splashing.

contain both air and liquid a splashing sound of cardiac rhythm may be audible but is simulated by sounds produced in large contiguous cavities as in *marked* gastric distension, pneumothorax and advanced phthisis.

ORGANIC HEART MURMURS.—Varieties.—Organic murmurs may be valvular, arterial, pericardial or pleuro-pericardial in origin.

Two kinds.

The valvular murmurs are of two kinds, (1) *regurgitant* and (2) *obstructive*—i.e. (1) those due to a leakage or back flow, and (2) those due to a narrowing of the valvular opening, a stenosis.

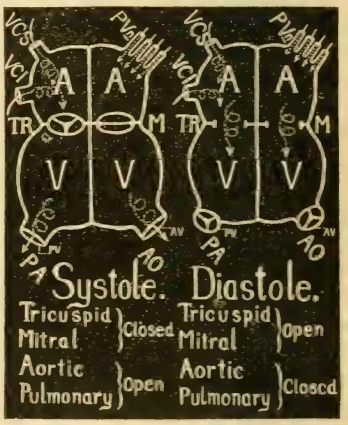


Fig. 82.—Position of the valves in systole and diastole.

Mode of Production.—*The heart must be regarded simply as*

Pump.

a double pump in constant action, or, as two double-chambered hearts cemented together and acting synchronously. *They may be shown diagrammatically as*

Relation to lungs.

two hearts with the lungs between them, for such is the plan of the circulation. *Pouring constantly into the upper chambers comes the blood from the greater and lesser circulations.* Into the right auricle the superior and inferior venæ cavæ pour their dark, impure, venous blood, while to the left, the four pulmonary

Course of blood.

veins bring a constant supply of bright, red, arterial blood, that has undergone purification in the pulmonary air-cells. *The blood passes from the auricles into the ventricles through the auriculo-ventricular valves*, the mitral on the left, the tricuspid on the right, *and is then forced by the ventricular contraction (systole) through the pulmonary valve on the right, and into the aorta upon the left.* As both sides of the heart contract

Blended sounds.

simultaneously, the sounds produced by valvular closure are ordinarily coincident and blended into one. *The "first sound" (lub) corresponds*

Ventricular contraction.

to the period of ventricular contraction and the closure of the mitral and tricuspid valves—i.e. **systole.** *The instant that this contraction is effected*

the emptied ventricles relax and the aortic and pulmonary valves, closing sharply to prevent backflow from the full arteries, produce by their sudden tension the "second sound" (dup), which initiates **"diastole."** *During this, the period of relaxation, the blood that has been accumulating in the auricles again pours down through the open mitral and tricuspid valves to fill the emptied ventricles, and, just before the next systole the auricles themselves contract vigorously and ventricular contraction immediately follows.*

Relaxation.

State of valves.

Auricular contraction.

Practical Application and Importance of a Mental Image.—*For the auscultator,* **systole** *is initiated by the first sound and* **diastole** *by*

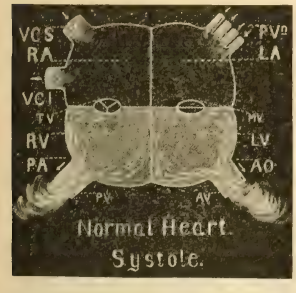


Fig. 83.—The Normal Heart in Systole. The full ventricles are contracting, the blood flows freely from them into the pulmonary artery and aorta; the mitral and tricuspid valves are tightly closed; the auricles are refilling. MV. Mitral valve. TV. Tricuspid valve. AV. Aortic valve. PV. Pulmonary valve. LA. Left auricle. RA. Right auricle. LV. Left ventricle. RV. Right ventricle. VCS. Vena cava superior. VCI. Vena cava inferior. PVn. Pulmonary veins. AO. Aorta. PA. Pulmonary artery.

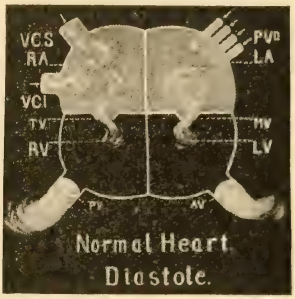


Fig. 84.—The Normal Heart in Diastole. The ventricular contraction has ceased, the aortic and pulmonary valves, tightly closed, are shutting off and supporting the blood column; the ventricles are filling from the open mitral and tricuspid orifices above. MV. Mitral valve. TV. Tricuspid valve. AV. Aortic valve. PV. Pulmonary valve. LA. Left auricle. RA. Right auricle. LV. Left ventricle. RV. Right ventricle. VCS. Vena cava superior. VCI. Vena cava inferior. PVn. Pulmonary veins.

the second. In the mind's eye he sees the cycle of events. Thus with the *first sound* he sees the *ventricles contracting,* the *aortic and pulmonary valves freely open* and the blood surging into the pulmonary artery and aorta. *With the second sound* he sees an exact reversal of conditions: *the aortic and pulmonary valves are tightly closed, the mitral and tricuspid freely open* and the blood is rushing through them from the auricles above to the ventricles below.

Phenomena of systole.

Of diastole.

Important Deductions.—*Any organic valvular murmur that immediately follows, replaces or modifies the normal first sound* (**systolic**

Inevitable conclusions.

murmur), *must arise either from leakage in the closed valves* (mitral and tricuspid) *or* from an *obstruction in those that should be freely open* (aortic and pulmonary). *Hence any organic endocardial systolic murmur must be due either to mitral or tricuspid regurgitation or to aortic or pulmonary obstruction* (stenosis). *In diastole the opposite conditions prevail; the mitral and tricuspid valves being open and the aortic and pulmonary valves closed. It must follow, that conversely, any murmur immediately following, modifying, or replacing the second sound* (diastolic) *is due to aortic or pulmonary leakage or to mitral or tricuspid obstruction. Nine out of ten murmurs have their origin in the left heart*—the hard working side.

Systolic murmurs.

Diastolic.

Rule of incidence.

Against the Stream.—It is at once evident that *if at any point there exist an impediment to the free flow of the blood, an increased strain is thrown upon the cardiac mechanism and a tendency to congestion or stasis is at once established.* Therefore, if any damming of the flow is present or any portion of the pumping machinery is defective, the bad effects will appear *chiefly* in those portions of the heart nearer the venous sources of the blood. *In short,* if one may use an old expression, *the bad effects of a cardiac lesion work backward against the blood stream.*

Logical sequence.

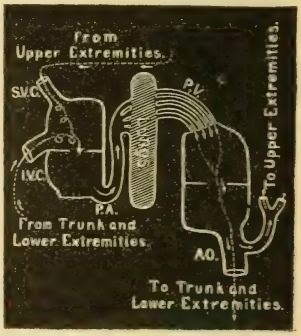


Fig. 85.—Right and Left Hearts S. V. C., I. V. C. Superior and inferior venæ cavæ. P. A. Pulmonary artery. P. V. Pulmonary veins. A O. Aorta.

Illustration.—*Interstitial nephritis* raises arterial blood pressure to an extraordinary degree and the left ventricle at once responds to the challenge of the narrowed arteries by increasing the strength of its contraction. The blood is then forced so vigorously into the aorta as to make the recoil or tension of the aortic valve unusually violent, and as a result there is an "*accentuation of the aortic second sound.*" Like every other muscle under unusual exercise, *the ventricle tends to hypertrophy* and to increase its strength: *the wear and tear of the aortic valve is also increased* and *an unusual and excessive pressure is exerted upon the mitral valve,* which *stands between* the overacting ventricle and the left auricle. *If,* as sometimes happens, *the mitral valve yields* to the pressure, a *backflow into the auricle* is at once established *and the flow of blood from the lungs is at once obstructed. The*

Heart response.

Tension.

Hypertrophy.

Relative insufficiency.

right ventricle is immediately called upon for increased action in order to relieve the congestion of the lungs. The blood is then forced so strongly into the pulmonary artery as to intensify the shock of its valvular closure and produce an "*accentuation of the pulmonary second sound.*" *The lungs may thus be placed between two fires through the continued regurgitation of blood from the front and the increased pressure from the overacting right ventricle behind.* A persistence of these conditions would tend to cause a *temporary or permanent leakage through the tricuspid valve, and the effects of back pressure are then manifested in the general venous circulation by congestion and edema, or, general anasarca.* Such a cycle of pathologic events is common enough in practice *though by no means invariable, and, realizing what marked local symptoms* may arise from even a slight chronic congestion of the brain, stomach, liver, intestines, kidneys and lungs, one can readily understand how easily in such cases valvular disease as a cause may be overlooked.

Pulmonary congestion.

Secondary tricuspid lesion.

Symptoms often misinterpreted.

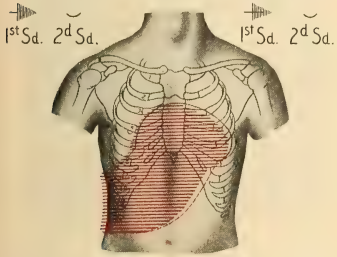


Fig. 86.—Mitral and Tricuspid Regurgitation. Percussion area—cardiac and hepatic. (*Ward cases.*)

The Causative Factors.—Practically all true valvular murmurs are due to stenosis or abnormal patency of one or more of the valvular openings and these conditions result from inflammation of the valves (endocarditis), chronic sclerosis (arterio-sclerosis), or from stretching of the valvular ring (relative insufficiency). The causative conditions are fully described, see pp. 213, 214, and a study of the causative factors makes clear the etiology of the actual lesions.

Endocarditis and sclerosis.

RHYTHM.—Organic Murmurs Occurring with Systole.—(First sound—period of ventricular contraction):—(a) *mitral regurgitation*, (b) *tricuspid regurgitation* (c) *aortic stenosis*, (d) *pulmonary stenosis* (rare).

Systolic murmurs are nine times in ten due either to mitral regurgitation or aortic stenosis, or, are purely accidental or anæmic.

Murmurs Heard in Diastole.—(Second sound—period of ventricular relaxation):—(a) *aortic regurgitation*, (b) *pulmonary regurgitation* (rare), (c) *mitral stenosis* (rarely purely diastolic, usually presystolic), (d) *tricuspid stenosis* (rarely).

Purely diastolic murmurs are almost invariably due to aortic regurgitation.

Murmurs Heard Immediately Before and Running Into the First Sound.—(**presystolic,** *auriculo-systolic.* Period of auricular contraction initiating systole):—(a) *mitral stenosis.* (b) *tricuspid stenosis.*

A presystolic murmur is almost invariably due to mitral stenosis.

Relative Frequency.—*Mitral regurgitation is by far the most common; mitral stenosis, aortic regurgitation, aortic stenosis and tricuspid regurgitation follow in order,* the remaining lesions being rare. *As to combined lesions,* double mitral and double aortic are most common, some placing the latter first in frequency, which is against the author's personal experience and would vary greatly in different clinics according to the predominant sex and age. *Mitral stenosis* is frequently overlooked and also misdiagnosed in connection with aortic regurgitation where the *false* presystolic or *Flint murmur* is frequently encountered. *True aortic stenosis is present only in a minority* of the cases presenting an aortic systolic murmur and, from the nature of the lesion, regurgitation must usually be associated with it. Thus all statistics so far presented are fallible and to some extent misleading.

Double lesions.

Variable.

Frequent errors.

Fig. 87.—Mitral and Tricuspid Regurgitation.—Heart in systole. Mitral and tricuspid valves both incompetent. *Result.*—Double systolic murmur, enlargement of both right and left chambers, pulsating jugulars, general venous congestion, edema, anasarca, etc. M v. Mitral valve. T v. Tricuspid valve. A v. Aortic valve. P v. Pulmonary valve. L A. Left auricle. R A. Right auricle. L V. Left ventricle. R V. Right ventricle. V. C. S. Vena cava superior. V. C. I. Vena cava inferior. P. Vn. Pulmonary veins. P. A. Pulmonary artery. A O. Aorta. Note:—Dilatation of the right heart and tricuspid leakage are much more common than was formerly supposed.

Sex and Age.—Mitral disease predominates in women, aortic in men. In children, females are chiefly affected and in 93% of Holt's series the lesion was mitral regurgitation, aortic leakage being extremely rare. As age advances aortic lesions increase in frequency, but the mitral valve still predominates.

The Differential Points.—To differentiate heart murmurs the following points are determined:—(a) *Time or rhythm.* (b) *Point of maximum clearness and intensity.* (c) *The direction and extent of transmission.* (d) *The quality of the abnormal sound.* (e) *The associated signs, viz.:*—arterial and venous pulsation, the radial pulse, increased area of cardiac dulness, accentuation of heart sounds, cyanosis, edema, etc.

All important.

To Determine the Rhythm or Time.—This should be positively determined by taking the apex beat or carotid pulse while listening to the murmur. *The radial pulse should not be used.* Use carotid.

Hypertrophy and Dilatation.—Figs. 72, 73, 74 show the variation in outline in these conditions; their influence upon heart sounds has been discussed and the peculiar differences in the apex beats. Simple hypertrophy is usually associated with chronic interstitial nephritis or arterio-sclerosis and simple dilatation represents the stage of incompensation in such lesions, or, some form of myocarditis. Disease associations.

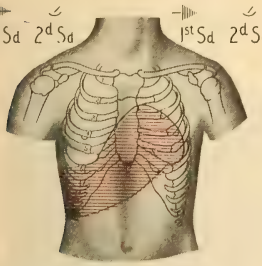


Fig. 88.—Mitral Regurgitation. Percussion area (cardiac and hepatic). (*Ward cases.*)

Characteristic Symptoms of the Systolic Valvular Murmurs.*—MITRAL REGURGITATION.—Time.—*Systolic.* **Quality.**—*Blowing*, of all degrees of intensity, occasionally musical. **Maximum Intensity.**—*At apex but sometimes heard only at the left inferior angle of the left scapula.* **Transmission.**—*To the left through the axillary space and just to the right of the edge of scapula between its spine and inferior angle.* **Associated Signs.**—*The pulmonary second sound is sharply accentuated: there is hypertrophy of the right heart*, and to a lesser degree of the left, *the apex being displaced to left and slightly downward.* Cardiac dulness is increased chiefly to the right; the cardiac area being well shown in the accompanying diagram (fig. 88). *If the lesion be fully compensated there are few symptoms aside from the physical signs;* if not, the *pulse* is usually irregular and of poor tension, *cyanosis* is present to a variable and often extreme degree, there is *passive pulmonary congestion or actual consolidation* and marked *dyspnœa or even orthopnœa.* The tricuspid may yield and with *loss of the pulmonary accentuation* the signs and secondary effects of right heart failure may ensue. *The œdema* varies with the degree of cardiac weakness, from slight swelling of the ankles or legs to general anasarca. *A patient is seldom so ill as to be beyond hope*, for no valvular lesion is more responsive to radical and rational treatment. This does not apply to those cases of sudden relative

Fig. 89.—Mitral Regurgitation. (*Sansom.*) Maximum intensity and transmission.

Compensation vs. Incompensation.

Œdema variable.

Hopeful prognosis.

* To be read in connection with "Compensation and Incompensation."

Bad cases.

insufficiency due to acute dilatation in which the prognosis is not good, death or chronic invalidism being a common sequel because of the absence of that gradual compensatory hypertrophy permitted by an endocarditis or arterio-sclerosis. Another extremely unfavorable mitral lesion is that which sometimes occurs as a terminal event in chronic interstitial nephritis.

AORTIC STENOSIS.—Time.—*Systolic.* **Quality.**—*Usually harsh*

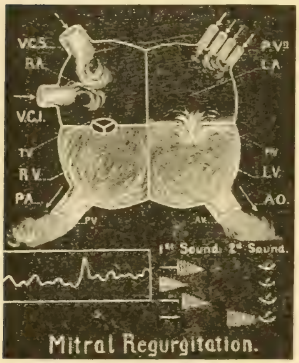


Fig. 90.—Mitral Regurgitation. Four varieties of the murmur of mitral regurgitation are shown graphically. The heart in systole, mitral leakage evident. The contracting ventricles are forcing the blood through the open aortic and pulmonary valves; the tricuspid, tightly closed, prevents regurgitation into right auricle. The leaky mitral allows backflow into the left auricle already filling from the pulmonary veins above. *Results.*—A systolic murmur, dilatation of left auricle, pulmonary congestion, and consequent enlargement of right ventricle. M V. Mitral valve. T V. Tricuspid valve. A V. Aortic valve. P V. Pulmonary valve. L A. Left auricle. R A. Right auricle. L V. Left ventricle. R V. Right ventricle. V. C. S. Vena cava superior. V. C. I. Vena cava inferior. P. Vn. Pulmonary veins. P A. Pulmonary artery. A O. Aorta.

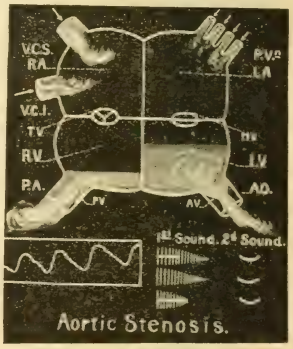


Fig. 91.—Aortic Stenosis.—A shygmographic tracing is shown, and three varieties of the aortic systolic murmur are represented graphically. Diagrammatic representation of the heart in systole, stenosis of the aortic valve being present; the mitral and tricuspid valves have closed; the right ventricle is nearly empty; the left ventricle is still more than half full of blood, because of the obstruction present at the aortic orifice. *Result.*—A systolic murmur in the aortic area; enlargement of left ventricle, etc. M V. Mitral valve. T V. Tricuspid valve. A V. Aortic valve. P V. Pulmonary valve. L A. Left auricle. R A. Right auricle. L V. Left ventricle. R V. Right ventricle. V. C. S. Vena cava superior. V. C. I. Vena cava inferior. P. Vn. Pulmonary veins. P. A. Pulmonary artery. A O. Aorta.

and blowing. **Maximum Intensity.**—*Right second intercostal space.* **Transmission.**—*Upward into carotid and subclavian, unless a very faint murmur.* May, if intense, be heard over the whole chest. **Associated Signs.**—*Hypertrophy or dilatation of the left ventricle.* Apex beat markedly displaced downward and to the left. *Pulse* usually

deliberate, *artery small and full between beats. Second sound in aortic area relatively diminished.*

Prognosis.—It is a lesion of long duration, but is not as common as is generally supposed, many systolic aortic murmurs being due to sclerosis of the aorta itself.

Common error.

Differential Diagnosis.—Its transmission alone is decisive save as regards sclerosis of the aorta *which shows an accentuated second sound.*

Sclerosis.

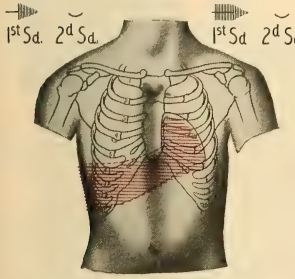


Fig. 92.—Aortic Stenosis. Percussion area. (*Ward cases.*)

TRICUSPID REGURGITATION.—Time.—*Systolic.* **Quality.**—*Blowing and usually soft.* **Maximum Intensity.**—*Over lower third of sternum* and occasionally chiefly to the right of its lower half. **Transmission.**—*May sometimes be heard over the whole right heart, and at times, if patient be recumbent, over the manubrium.* **Associated Signs.**—*Hypertrophy or dilatation of right heart. Diminished accentuation or loss of pulmonary second sound, distended or directly pulsating jugular veins.* In bad cases, or as the lesion advances, it may, by back pressure through the general venous system, lead to extreme chronic congestion of the liver, spleen, stomach and intestines, edema of the lower extremities and, general anasarca. *It is frequently secondary to a mitral lesion*, but may be induced by heavy strains, particularly if the myocardium be primarily diseased. It is not uncommon in heavy drinking teamsters or laborers, especially brewery employes, "beer-driver's heart," and indeed *is more common than is usually stated.*

Systolic venous pulse.

Primary vs. Secondary.

Common.

Fig. 93.—Aortic Stenosis. Transmission.

Prognosis.—Poor if the disease is primary but it often disappears entirely under treatment if the insufficiency is secondary and relative.

Differential Diagnosis.—Is often confounded with mitral regurgitation but its characteristic location and transmission, the pulmonary second sound diminution distinguish it from everything except pulmonary stenosis, an excessively rare, usually congenital, lesion having its maximum at the second left intercostal space.

Diagnosis easy.

PULMONARY STENOSIS.—*Systolic murmurs in the pulmonary area are almost invariably functional. The organic murmur is extremely*

Rarely organic.

Rare.

rare and usually congenital. If present it is commonly associated with other congenital defects, such as a patent foramen ovale or perforate interventricular septum. The *differential* diagnosis is at times extremely difficult, sometimes quite impossible. The typical signs are the following:—

Fig. 94.—Pulmonary Stenosis in Adults.—(*Sansom.*) Shaded area represents field of maximum intensity.

Fig. 95.—Pulmonary Stenosis. *Maximum point and direction of transmission.

Quality.—*Rough, harsh murmur.* **Time.**—*Systolic.* **Maximum Intensity.**—*At second or third left interspace.* **Transmission.**—*May be heard over whole right heart, but is usually markedly transmitted from pulmonary area upwards and outward toward the clavicle. It is not transmitted along the aorta and into the carotids.* **Associated Signs.**—*Diminished pulmonary second sound. Hypertrophy of right heart. Cyanosis* is easily produced by cough or exertion and may be permanent. It will certainly be so if a patent foramen ovale co-exist with the pulmonary stenosis. Its victims usually die young of pulmonary tuberculosis.

Important transmission.

Blue children.

Characteristic Symptoms of the Diastolic Valvular Murmurs.—The diastolic valvular murmurs are, theoretically, mitral stenosis, aortic regurgitation, tricuspid stenosis and pulmonary regurgitation. *An exception must be made in the case of stenosis of the mitral or tricuspid orifice.* Such murmurs are rarely diastolic, possibly because the flow of blood from auricle to ventricle during diastole is not sufficiently forcible to produce a murmur until just before systole, when the ventricular suction is reinforced by auricular contraction. For this reason the rhythm of mitral or tricuspid murmurs due to stenosis is presystolic or auriculosystolic—i.e. *beginning late in the diastolic period, they are heard just before, and cease abruptly at, the first sound.*

Fig. 96.—Mitral Stenosis.—(*Sansom-Bramwell.*) *Point of maximum intensity and transmission.

Fig. 97.—Mitral Stenosis.—(*Sansom.*) *Upper shading*, Area of audibility of simulated doubling of second sound. *Lower shading*, Area of murmur transmission.

Presystolic murmurs.

MITRAL STENOSIS.—Quality.—*Harsh, vibratory or blubbering murmur terminating abruptly in marked crescendo.* **Time.**—*Presystolic.*

Increasing in intensity as it runs into a sharply accentuated first sound. Rarely diastolic and still more rarely divided. **Point of Maximum Intensity.**—*At or just* within the beat. **Transmission.**—*Limited to the mitral area and usually very sharply defined.* **Associated Signs.**—*Cyanosis present to some degree* but often slight. *Accentuated pulmonary second and mitral first sounds.* It is frequently accompanied by a systolic mitral murmur (regurgitation). *Marked hypertrophy of right ventricle,* i.e. percussion dulness extends to right of normal boundaries. **Pulse.**—*Small, rapid, irritable and irregular. Thrill usually present,* felt best at or near point of maximum intensity of murmur. A diagnosis may often be made by palpation, *a presystolic thrill in the mitral area being pathognomonic of mitral stenosis.* Second sound frequently doubled.

Diagnosis sign.

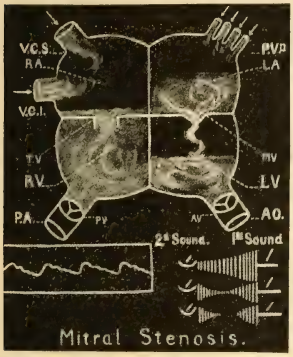


Fig. 98.—Graphic Representation of Three Varieties of the Murmur of Mitral Obstruction. (With Sphygmogram.) Heart at moment of auricular contraction immediately before systole (presystole); mitral obstruction evident; aortic and pulmonary valves closed; tricuspid freely opened; *right* auricle nearly empty; right ventricle filled; *left auricle but partly emptied; left ventricle barely half full. Result.*—Presystolic or diastolic murmur, dilatation of left auricle, congestion of lungs, consecutive enlargement of right heart. M V. Mitral valve. T V. Tricuspid valve. A V. Aortic valve. P V. Pulmonary valve. L A. Left auricle. R A. Right auricle. L V. Left ventricle. R V. Right ventricle. V. C. S. Vena cava superior. V. C. I. Vena cava inferior. P. Vn. Pulmonary veins. P. A. Pulmonary artery. A O. Aorta.

Sources of Error.—The diastolic murmur of mitral stenosis may be confounded with a rare form of aortic regurgitant murmur that is distinctly heard at the apex. The presystolic murmur is often variable or intermittent, may be heard only in either the standing or the recumbent position, and may be entirely obliterated by pressing too heavily with the bell of the stethoscope. *The sharp flapping first sound is usually so strikingly characteristic as to suggest the presence of mitral stenosis even when no murmur is present.*

Attitude.

Stethoscopic pressure.

Almost pathognomonic.

AORTIC REGURGITATION.—Quality.—*A blowing murmur of variable pitch and intensity, usually somewhat prolonged, occasionally very short, perhaps musical, which may entirely or partly replace or but slightly modify the second sound.* **Time.**—*Diastolic.* (Commencing with or immediately following the second sound.) **Maximum Intensity.**—*At aortic area* (second right interspace), *third left interspace, or at ensi-*

form. It is increased by raising the arms to a vertical position and by exertion and very rarely this murmur is heard only at the apex. **Transmission.**—Along lines shown in fig. 101, i.e., from *second right interspace to second left or third left cartilage, down left edge of sternum;* is frequently *heard at apex and in the carotids.* **Associated Signs.**—*Pallor, visible jerking pulsation of peripheral vessels*—(i.e., temporal, carotid,

Rich in signs.

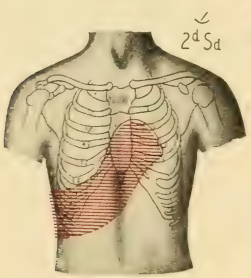


Fig. 99.—Mitral Stenosis. Percussion area. (*Ward cases.*)

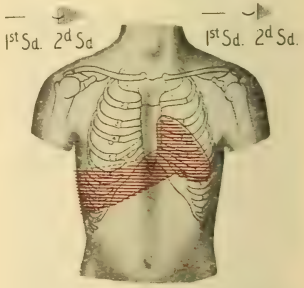


Fig. 100.—Aortic Regurgitation. Percussion area. (*From ward cases.*)

subclavian, axillary, brachial, radial, etc.). *Capillary pulse: second sound diminished or lost in carotids:* sometimes a pseudo-mitral stenosis (Flint) murmur. *Capillary pulse:*—If the nails be examined closely, if a glass slide be pressed upon the lips, or if the skin be reddened by friction, a rhythmic flush may be seen. **Radial Pulse.**—*A jerking, throbbing, slapping pulse* (the "Corrigan" or "water-hammer" pulse) which may be felt in shaking hands or in grasping the tips of the fingers or toes. In order best to appreciate the lack of sustained tension it is well to raise the arm and grasp the wrist with the hand in such a way that the ball of the examiner's thumb overlies the radial artery, the finger and thumb meeting on the back of the hand. **Cardiac Area.**—The cardiac area is increased downward and to the left because of left ventricular hypertrophy. *Pistol-Shot sound in femorals.* This curious sharp shock-like sound may be heard in the femoral arteries in marked cases. *Dizziness, fainting, palpitation, cardiac pain, and neurasthenic symptoms are common in this lesion.* The ophthalmoscope may show *jerking retinal arteries.* The second aortic sound is markedly diminished and usually

Useful manœuvre.

Fig. 101.—Aortic Regurgitation. * * * maximum intensity direction of transmission.

Ophthalmoscopic signs.

lost in carotids. *Aortic regurgitation is frequently associated with aortic stenosis.*

General Symptoms and Comment.—As in other lesions, no marked subjective symptoms may be present while compensation is maintained but, more often than in them, a dull pain or sense of oppression in the cardiac area or sharper neuralgic pains referred to the neck, shoulder, and especially the left shoulder and arm, or even true angina pectoris, may be noted, Excitement and physical exertion may initiate or provoke recurrence of these symptoms. Vertigo, tinnitus aurium, throbbing headaches, syncope, subjective or true dyspnœa, and palpitation may be present. The patient is usually nervous, high strung, and, if he knows his condition, apprehensive and easily depressed. The mental state is often that of neurasthenia and in rare instances actual melancholia is observed. Sleep is often disturbed by dreams or an actual insomnia exists frequently aggravated by the auto-audible beating of the carotids or other vessels, of which these patients often complain. In the stage of incompensation there is little tendency to general anasarca or marked pulmonary congestion unless, as frequently happens, there is a co-existent or secondary mitral lesion. Edema of the ankles is common, however, and dyspnœa, palpitation, oppression, etc., tell the tale of cardiac weakness.

Pain.

Vertigo, etc.

Nervous state.

Edema late or absent.

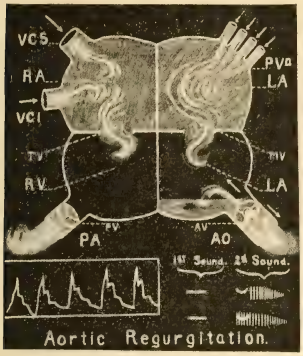


Fig. 102.—Graphic Representation of Murmur. Two Varieties of Aortic Diastolic Murmur Shown Graphically. (With Sphygmogram.) The heart is shown in diastole, aortic leakage being evident. The blood has just been projected into the aorta and pulmonary artery by the ventricular contraction. The pulmonary valve tightly closed maintains the blood column, but through the leaky aortic valve a regurgitant current meets the stream descending from above through the open mitral valve. *Results.*—A diastolic murmur, dilatation and hypertrophy of left ventricle, a slapping, low-tension pulse. M V. Mitral valve. T V. Tricuspid valve. A V. Aortic valve. P V. Pulmonary valve. L A. Left auricle. R A. Right auricle. L A. Left ventricle. R V. Right ventricle. V C S. Vena cava superior. V C I. Vena cava inferior. P Vn. Pulmonary veins. P A. Pulmonary artery. A O. Aorta.

TRICUSPID STENOSIS.—*Rare, usually congenital.* **Quality and Time.**—*Exactly like mitral stenosis.* **Point of Maximum Intensity.**—*Lower sternum, chiefly along right border.* **Transmission.**—*Slight, along right sternal border.* **Associated Signs.**—Marked cyanosis. Distension of jugulars. **Percussion Area.**—That of right auricular dilatation.

PULMONARY REGURGITATION.—*Rare, usually congenital.* **Quality and Time.**—*Exactly like murmur of aortic regurgitation.* **Point of Maximum Intensity.**—*Second left interspace.* **Transmission.**—*Over sternum and down its left edge.* Differentiated from aortic regurgitation by the percussion area, which is that of a hypertrophied right ventricle, and by the *associated signs*. These are cyanosis and overdistension of jugulars. *In this lesion the second aortic sound is clearly heard.* Marked jerky pulsation of peripheral arteries, the "Corrigan pulse," and the capillary pulse are wanting.

Fig. 103.—Aortic Regurgitation. Aneurismal dilatation. Mitral leakage.

Rarer Murmurs.—As a matter of fact, *the organic pulmonary murmurs and that of tricuspid stenosis are so rare in adults that one has practically to consider only mitral, tricuspid and aortic regurgitation and mitral and aortic stenosis.* As previously stated *a systolic murmur* is due, nine times out of ten, to mitral regurgitation or aortic stenosis, and as one is heard best at the apex and transmitted to the axilla and back, and the other is best heard at the aortic cartilage and transmitted upward, no confusion need occur. *The purely diastolic murmur* is almost certainly due to aortic regurgitation and its peculiar transmission and the associated signs make it quite unmistakable. *The chief source of error lies in a failure to take the time of every murmur by the carotid beats*, carefully note its maximum intensity and transmission and, in the case of two murmurs like in rhythm, the pitch and quality.

Ready differentiation.

Timing murmurs.

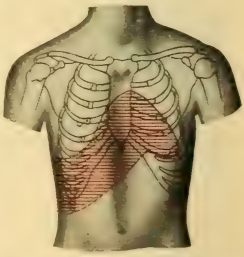


Fig. 104.—Aortic and Mitral Regurgitation. Percussion area. (*Ward cases.*)

Associated Murmurs.—Any combination of murmurs may be encountered though in most instances a post mortem greatly reduces the excessive number of presumptive lesions sometimes reported.

CONGENITAL HEART DISEASE (morbus cœruleus.).—The "little victim" is not always blue, for certain defects such as those of the auricular septum may exist without cyanosis. On the other hand a baby

may in exceptional cases be decidedly blue with no evidence of heart disease, as in a case known to the author in which a large mass of adenoid tissue in the *naso pharynx* was accountable for the condition which disappeared at once after operation. The rule holds however that *an active infant or ambulant child that is extremely cyanotic* is the victim of congenital heart disease and in most instances *loud*, harsh, often musical *murmurs* are heard, often without the thrills or characteristic changes in cardiac outline pertaining to the ordinary heart lesions which they may simulate. Clubbing of the fingers and toes and dyspnœa frequently co-exist.

Usual inference.

Cardinal points.

The lesions aside from those already discussed may be thus epitomized:—**Auricular septum defects**—no definite symptoms. **Ventricular defects**—simulate mitral regurgitation but lack symptoms of pulmonary engorgement. Not to be positively diagnosed.

Persistent Ductus Arteriosus.—Post systolic murmur *continuing beyond the second sound* and transmitted slightly into carotids; radial pulse smaller in deep inspiration. Aneurismal dilatation of the duct may produce paralysis of vocal cords through recurrent laryngeal nerve, There may be late systolic thrill, dyspnœa and cyanosis and slight second pulmonary accentuation.

Persistent Aortic Isthmus.—Arteries and pulse of upper extremity (branches from the arch) are large. Those of the lower extremity and trunk small, the pulse being markedly delayed. The left ventricle is hypertrophied and there are visible collateral arterial communications (e.g. internal mammary—sup. and inf. epigastric, dorsalis scapulæ and posterior intercostals, etc.) which may yield systolic murmurs and thrills.

Comment.—Unfortunately these lesions, sufficiently vague and indeterminate in themselves are usually combined and seldom permit a specific localizing diagnosis *The murmurs* are systolic or post systolic save in pulmonary regurgitation and are usually loudest at the base. *Pulmonary lesions* cause a marked enlargement of the right heart and a striking diminution of the pulmonary second sound with marked thrill in stenosis. *Ventricular septum defect* may lack both thrill and hypertrophy. *Patent ductus arteriosus* may show marked thrill and no hypertrophy. *A persistent aortic isthmus* is plainly indicated.

Other Congenital Anomalies.—The heart or the great vessels may be transposed, consist of one, two, or three chambers, through complete septal defects, or, the heart may be placed in the neck, abdomen, or, without the chest cavity.

COMPENSATION AND INCOMPENSATION.—A clear under-

Must be understood.

standing of these two factors as affecting diagnosis, prognosis, and treatment is absolutely essential, and a lack of it leads to sins of commission no less than omission. Owing to the wonderful interaction of the regulating mechanisms controlling the blood vessels and the heart almost every form of heart defect may be so compensated for varying periods as to equalize the circulation and minimize symptoms. A clinician recognizes three distinct phases in the course of any heart lesion: (1) *perfect compensation*, (2) *impaired compensation*, (3) *lost compensation.*

Three stages.

As regards the four chief varieties of valvular lesions, namely, mitral regurgitation and stenosis, and aortic regurgitation and stenosis, *the first stage* may show nothing more than the peculiar murmurs, the characteristic change in the cardiac outline, which indeed may be slight for a long period, and certain characteristics in connection with the pulse or general peripheral circulation. This statement is equally true of certain forms of myocarditis with intermittent *relative valvular* insufficiency and indeed applies to a greater or less extent to all lesions. Sins of commission occur in connection with this state and consist of meddlesome interference in the way of treatment and the creation of unnecessary alarm through irrational and terrifying statements as to prognosis. *Such hearts need little or no treatment but do need watching from time to time.*

Slight symptoms.

Error frequent.

(2). **Impaired Compensation.**—This stage is the one most commonly encountered and the sins committed in connection with it are chiefly those of omission due to a failure to recognize and interpret obscure manifestations of the condition. In many lesions these symptoms are actually obtrusive; for example, the victim of *aortic regurgitation* complains of dull pain and oppression in the precordium or sharper neuralgic pains radiating to the neck, left shoulder and down the left arm, or, he has some subjective or objective dyspnœa, attacks of *pronounced* angina pectoris, vertigo, headache, insomnia, probably an increase of the nervous irritability so common in these cases, or, signs of secondary mitral lesions may appear. In *mitral stenosis* the dyspnœa may be marked, especially on exertion, the pulse becomes rapid, more markedly irregular and unequal, pulmonary congestion and blood streaked sputum may be evident and this may even be reflected in the condition of the kidney, *stomach*, liver and spleen. In *mitral regurgitation* much the same symptoms are evident and if the right heart is markedly weakened or *tricuspid regurgitation* is established, there is marked diminution of the pulmonary second sound, direct pulsation of the extended jugulars, pulmonary congestion, enlarge-

Frank or vague.

Pain.

Dyspnœa.

Secondary lesions, etc.

ment of the spleen and liver, indigestion, flatulence and in short general, visceral, passive congestion. *These symptoms are sufficiently striking and to them are added in any case an extention of the cardiac borders due to a dilatation which may be manifested by the substitution of a weakened heart impulse for that of hypertrophy, and oftentimes, by a change in the quality of the murmur.* In *aortic lesions* edema is likely to be absent or slight so long as the mitral holds fast. In *mitral lesions* edema of the lower extremities *may* be present with even slight degrees of incompensation. In the later stages general anasarca may appear. *These are plain straightforward symptoms, each with its clear meaning, but it is quite otherwise when one deals with the slighter forms of such incompensation* and still more so in relation to the dilatation and slight incompensation associated with *chronic myocarditis;* cases which frequently present no murmur until rest and cardiac stimulants have renewed the tonus of the heart muscle and narrowed the valvular ring.

Heart borders.

Edema.

Obscure types.

The nervous system is peculiarly affected in many cases of slight incompensation. *Some of the most typical cases of neurasthenia ever seen by the author have been associated with a silent or almost silent leakage or stenosis of the mitral valve, and indeed several were associated with unrecognized aortic aneurism.* Nothing is commoner in these cases than nervous irritability, drowsiness or insomnia, lack of concentration and ability to perform sustained mental or physical work, while numbness and tingling of the extremities are frequent. Again and again these cases have been associated with only slight increase of the cardiac area, enfeebled or somewhat fœtal heart sounds, and most often, *markedly diminished or absent mitral first sound.* **The skin** may be the seat of persistent pruritus or eczema and very often a very slight grade of edema may be present, limited usually to the lower extremities, bilateral, and often most marked over the surface of the tibia.

Mistaken for neurasthenia.

The Lungs.—Persistent dry cough, slight or even marked congestion of the bases, asthmatic seizures, diffuse bronchitis, moderate grades of emphysema and persistent pharyngitis and laryngitis may divert attention from an underlying incompensation, which is either causative, or a factor in the severity and continuance of pulmonary symptoms.

Treated as pulmonary lesions.

The Gastro-intestinal Tract.—Indigestion and persistent constipation together with recurrent attacks of acute gastric and hepatic disturbance and hemorrhoids are frequently associated with inefficient heart action. The author firmly believes that many of the results

Mistaken for gastric disease.

obtained by the rest cure in neurasthenia may be attributed to the good effect upon the *stomach and heart with the resulting improvement in nutrition.* Certainly under mental and physical rest all of the symptoms described promptly disappear in all minor degrees of incompensation.

(3). **Complete Incompensation.**—*This term should be applied to those cases in which the heart muscle has completely lost its recuperative quality* as is seen in the terminal stages of all chronic heart affections that terminate gradually, or by sudden though not immediately fatal rupture of compensation. The best examples are seen in terminal cases of coronary sclerosis, fatty heart and chronic myocarditis in general. So also in mitral lesions there comes a time when the heart that has alone or with assistance again and again recovered itself finally yields and resists all therapeutic measures. In such terminal and "*irrecoverable*" cases the orthopnœic patient often rolls the head aimlessly from side to side and wears a peculiarly listless yet distressed and hopeless expression. The term is frequently erroneously applied to cases of very marked and extreme cardiac weakness and especially to that of mitral regurgitation or stenosis associated with secondary tricuspid leakage and general anasarca. *In mitral regurgitation especially, the assumption of terminal incompensation is seldom justified as a primary assumption, for there is no cardiac lesion in which proper treatment can do so much, however extreme may be the manifestations.* Indeed in the case of all heart lesions it is only after trying and failing that surrender is justifiable on the part of the physician.

Hopeless cases.

Extreme incompensation sometimes temporary.

When to surrender.

Fig. 105.—Hopeless incompensation. Attitude frequently assumed. Triangular area represents superficial cardiac dulness.

Relative Insufficiencies.—The secondary dilatation of the valvular rings, such as occurs in the tricuspid following prolonged mitral lesion or as a result of myocarditis and severe physical strain, or in the mitral as secondary to aortic lesions, chronic myocarditis or the terminal stage

of an interstitial nephritis the prognosis is variable. In such cases under proper treatment the tricuspid usually recovers its function in whole or in part, as does the mitral to a less degree and with less con-

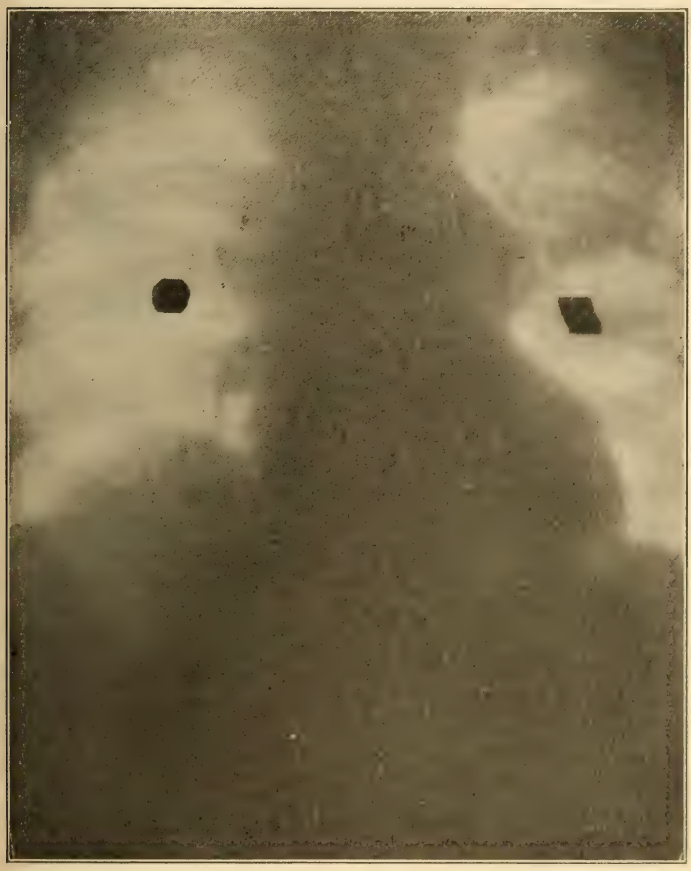


Fig. 106—Aneurism involving ascending and transverse portions of the arch, coincident aortic regurgitation. Symptoms of aortic regurgitation only at time photograph was taken. Later those of aneurism became evident.

stancy. *The left heart cases associated with sudden rupture* of compensation *through severe strain do badly if the myocardium is not sound, and often terminate quickly and fatally.* So also the mitral insuffi-

Sudden rupture

Interstitial nephritis.

ciency so often encountered in chronic interstitial nephritis will not respond to treatment with anything like the promptness usually noted in connection with this valvular lesion, and, moreover, the patients of this type are often irritable, restless, difficult to control and quite unlike the ordinarily phlegmatic, cheerful mitral-regurgitant patient.

THORACIC ANEURISM.—*A true aneurism represents a local dilatation of an artery due to the weakening effect of a chronic degenerative arteritis.*

Etiology.—Any factors that lead to arterial degeneration on the one hand and abnormally great vascular tension upon the other may produce aneurism. As is often said, the victim is usually one who has worshipped at the shrine of Venus, Bacchus or Vulcan, and to these the author would add Mammon if not Minerva. *Syphilis unquestionably stands first as a cause and is responsible for a large majority of the cases. Intemperance* in eating or drinking, *physical and mental overwork or overstrain* act as contributing causes, the first symptom in many cases dating from sudden, severe or prolonged muscular effort. Several cases coming under the author's observation have dated their symptoms from severe falls or railway accident. It is quite possible that a congenital weakness of the vessels is a factor in the aneurisms of the young but in all these cases it is difficult to believe that we can exclude a primary inherited degenerative process due to one of the factors originally mentioned. *The disease is much more common than is generally supposed and is frequently the cause of sudden death referred (without autopsy) to other conditions.*

Devotees.

Traumatism.

Heredity.

Difficulties Encountered in Diagnosis.—The use of the fluoroscope and the X-Ray photograph has added greatly to our diagnostic resources and made it possible to recognize aneurism in its earlier stages. *Otherwise no disease is more easily recognized in the presence of its classical symptoms, nor more frequently overlooked when these are absent.* The old clinical divisions are (a). *Aneurism with signs and symptoms.* (b). *Aneurism with symptoms but no signs.* (c). *Aneurism with neither symptoms nor signs.* It should be remembered that (a). Classical symptoms develop only in certain cases and then usually when the terminal stage is reached. (b). That large aneurisms may exist without them. (c). That death from aneurism is frequently unsuspected both ante and post mortem in the absence of an autopsy. (d). That *ordinary diagnostic resources* are therefore unsatisfactory and inefficient and (e). *That the fluoroscopic picture or X-Ray photograph offers usually the only means of early diagnosis at our command.*

Valuable aid.

Old divisions.

Important.

Statistics of Aneurism.—Roughly speaking three-fourths of all aneurism are aortic, and nineteen-twentieths of these are found in the thoracics aorta; of these 90% are saccular; from 80–90% occur in the male, and 50% occur between the ages of 35 and 50. The people whose habits most largely represent intemperance in food and drink and occupations which involve the maximum of intemperance and liability to syphilitic infection furnish the chief examples to the incidence and mortality of the disease.

Favorite Sites.—The root of the aorta, the junction of it ascending and transverse portions, and the descending arch represent the chief points of attack in the frequency indicated by their order.

Termination.—The peculiar situation of these tumors with a reference to adjacent and related structures readily explains their symptomatology and termination. *Death occurs suddenly in almost every instance because of rupture of the sac.* The blood may pass into the pericardium or adjacent pulmonary artery, into the superior vena cava, the esophagus, any one of the four heart chambers, the lung itself, the mediastinum, or, externally, in aneurisms causing pressure necrosis. *As regards relative frequency the pericardium, pulmonary artery, and right auricle head the list in the order given.*

Sudden death.

Symptoms.—A pulsating gradually enlarging tumor within a limited space filled with important anatomical structures means a predominance of *pressure symptoms*, which may be tabulated as follows:—*Esophagus*, dysphagia. *Trachea:*—brazen cough (gander cough), dyspnœa, stridor, bronchorrhœa and hemoptysis (if not from sac itself). *Root of the lung, and the pleura:—Symptoms suggesting phthisis*, pulmonary collapse, pleurisy, etc. *Pericardium:*—Pericarditis. *Chest wall:*—Localized, dull pain. *Nerve trunks:*—Neuralgic pains, paroxysmal and intermittent. *Pulmonary artery*, systolic pulmonary murmur, dilated right heart. *Sympathetic fibres:*—Dilated or contracted pupil, unilateral sweating or pallor. *Cardiac plexus*, anginal attacks. *Superior vena cava:*—Edema of upper extremity, cyanosis. *Thoracic duc:*—Marasmus. Vagus:—Dyspepsia, nausea, vomiting, dyspnœa, hiccough. *Phrenic:*—Unilateral diaphragm paralysis. *Recurren: laryngeal:*—Hoarseness, aphonia, spasm or paralysis left cord, *paroxysmal dyspnœa. It must not be forgotten that any or all of these symptoms may be caused by mediastinal growths other than aneurisms.*

Predominant pressure symptoms.

May be absent.

PHYSICAL SIGNS OF ANEURISM.—Inspection.—This must be both direct and tangential and one seeks primarily to discover *local bulging* or pulsation. The region of the manubrium and the

back between the left scapula and the spine should be examined with special care.

The Primary Signs Are:—(a). *Abnormal pulsation* usually in the locality mentioned, but variable in position, degree and extent;

Fig. 107.—Diffuse dilatation of aorta, aortic stenosis and regurgitation. Dilated vessels show here as a faint shadow.

if typical, heaving and expansile. (b). Rarely, *a visible tumor yielding expansile pulsation* and covered by tissues which may be normal, tense and shiny, congested or even necrotic.

Secondary Signs.—(a). *The peripheral signs of an associated aortic*

regurgitation may be present. (b). *The apex beat may indicate left ventricular hypertrophy*, a condition commonly but not invariably associated with thoracic aneurism. (c). *Signs of sclerosis in the peripheral arteries.* (d). *Cyanosis, localized edema and unequal pupils, vasomotor symptoms such as unilateral pallor, congestion or sweating.* (e). *Stridor, visible dyspnœa.* (f). *Paralysis of the vocal cords* associated with hoarseness or aphonia. (g). *Brazen cough.*

Palpation.—*Expansile pulsation and thrill and the so-called disastolic shock* are chiefly to be sought. In certain aneurisms involving the transverse portion of the arch, one may find the "*tracheal tug*" first described by Oliver. To obtain this, the cricoid cartilage is grasped by the thumb and finger of the observer as the head of the patient is tipped slightly backward, upward traction is then made and a tugging sensation may be felt with each cardiac impulse.* The observer is often misled by the pulsation of vessels under the finger, particularly if aortic incompetence exists, and, by *checked* respiratory movements. *Furthermore, it is undoubtedly present in many healthy persons, and in those who have residual pleural adhesions and certainly absent in a very large proportion of the cases of true aneurism.*

Tracheal tug over-rated.

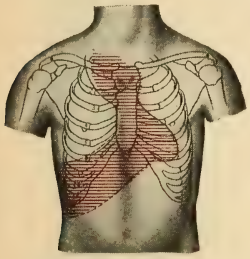


Fig. 108.—Aneurism of the Aorta. Percussion area in a case presenting typical signs. (Ascending portion.)

The Pulse.—Aside from the signs of a coincident aortic regurgitation the pulse yields information of real value in many cases. The observer should carefully compare the beat in the two radials *and carotids* and note any delay or inequality. Resulting as they do either from the pressure of the sac or deformed arterial outlets in the portions involved, such differences furnish excellent corroborative evidence and sometimes assist in localizing aneurismal tumors. (See p. 171.)

Important.

Percussion.—*In advanced and well defined cases* percussion may furnish direct evidence in the form of marked dulness over aneurismal areas, *but in the earlier cases it is deceptive and misleading. Auscultatory percussion* is usually more valuable than simple percussion. Student and practitioner alike should be thoroughly familiar with the normal variations in the peculiar percussion note yielded by the manubrium sterni.

Often useless.

* From the careful study of a considerable number of cases the author believes this to be a greatly over-rated symptom.

Important, not constant.

Auscultation.—A *systolic* or more rarely a *diastolic murmur*, or both, may be heard but are due in most instances to associated valvular disease, this being particularly true of the diastolic murmur, a systolic bruit often harsh, vibrant and associated with palpable thrill being not uncommonly produced by the sac itself. Pressure upon the pulmonary artery may also account for the murmur heard. The systolic murmurs are often widely transmitted along the vessels and a localized systolic heard at the left back is an important sign of aneurism of the descending thoracic aorta. *The most important single sign, the author believes to be a distinct ringing, metallic, second sound, heard not over the aortic valve itself, but over the sac.* The maximum intensity of the true sac murmurs may occupy the same suggestive site.

Valuable suggestive sign.

Subjective Symptoms.—*Dyspnœa, precordial oppression and pain*

Fig. 109.—Aortic Aneurism (Ascending Portion of Arch). Unusual percussion area. +. Only point of pulsation. Had taken out insurance within two years.

Fig. 110.—Aortic Aneurism. Same case as figure 109, three months later.

are the chief subjective signs and of these, pain is by far the most important. *Theoretically*, every large aneurism should be accompanied by *severe* pain, *actually* a large number go to a fatal termination without it. When present it may be localized, diffuse or referred, shooting, cutting, or more commonly dull or gnawing. It is often misinterpreted, regarded as neuralgic, and so treated.

May long be absent.

Special Symptoms According to Site of Lesion.—*Aneurism of the first portion of the arch.* Any statements made under this heading are liable to exception, but in general it may be said that if the first portion of the arch is involved a pulsation or rarely an expansile tumor

is likely to appear at or about the second right interspace, which tends to extend its area of dulness outward and upward. In exceptional instances the mass passes downwards. Attacks of angina pectoris and severe palpitation are common in this connection. The aortic valves are likely to be involved and symptoms of aortic regurgitation and a double murmur are often present, associated with systolic thrill and marked diastolic shock. Extension of the growth tends to cause localized edema, cyanosis, vaso-motor symptoms and possibly pleural or even pericardial inflammation.

Dulness.

Pain.

Thrills and murmurs.

Aneurism of the Transverse Portion of the Arch.—The common symptoms are dulness and pulsation over and about the manubrium and in the *episternal notch, tracheal tug in some cases*, paralysis of the vocal cord with whispering voice and brazen cough, marked general pressure symptoms, *pain*, variable (less severe than in aneurism of the first portion), *systolic bruit, thrill*, diastolic shock (often lacking), pulse variations (often marked), dyspnœa and precordial pressure. *Pseudo-asthmatic* seizures are common and misleading in connection with this form and the author has known of several such instances in which for a long time treatment had been unsuccessfully and mistakenly employed.

Fig. 111.—Aneurism. (Aortic). This man took out life insurance but a short time before photograph was taken.

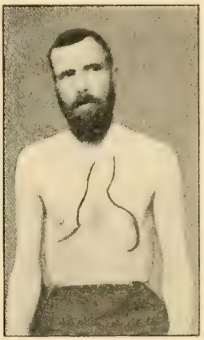


Fig. 112.—Aneurism. Man well nourished and healthy in appearance.

Symptoms often marked.

Pseudo asthma.

Aneurism of the Descending Portion of the Arch.—In this lesion the symptoms are likely to be present posteriorly rather than anteriorly. They may be slight or absent; may include bruit, percussion dulness, superficial posterior pulsation, or, actual tumor with

Obscure.

perhaps external perforation, and are commonly accompanied by a considerable degree of posterior dulness and gnawing pain.*

General Considerations and Differential Diagnosis.—The last 20 cases of thoracic aneurism coming under the author's attention have been subjected in each instance to the X-Ray, and without it fully 50% would have gone unrecognized. By reference to illustrations on pages 208, 209, one will see that advanced cases terminating fatally within a few months, presented few or none of the typical signs of the disease. Many had been treated for various ailments, utterly unrelated to the true cause of the trouble, and neuralgia, asthma, dyspepsia, disease of the spine and pulmonary tuberculosis were amongst the diseases erroneously diagnosed. It would seem that our ideas in regard to the symptomatology of thoracic aneurism need revision and that our diagnostic methods should be reinforced by the findings of the X-Ray.†

Value of X-Ray.

Early diagnosis rare.

Differential Diagnosis.—*Mediastinal abscess.* The rapid development with symptoms of suppuration and inflammation and the absence of aneurismal symptoms other than pressure symptoms should be sufficient to exclude this lesion. *Pulmonary fibrosis.* Cases of fibroid phthisis or any case involving the retraction of lung may give rise to suggestive pulsation in aneurismal areas, but ordinary methods should be sufficient to establish the actual conditions present and be reinforced by a fluoroscopic inspection of the arch. *Pulmonary tuberculosis* with large cavity adjacent to the aneurismal areas may produce pulsation and certain other aneurismal signs, but the history, sputa, breath sounds, etc., should make error impossible. **Anæmic Pulsation** should be readily differentiated by rational methods. *Aortic regurgitation is frequently associated with aneurismal* dilatation both dynamic and true; if unassociated it should offer little difficulty. *Malignant growths in the mediastinum.* When these are associated with evidences of malignant disease elsewhere in the body they offer little difficulty, but when not so associated they become most difficult to differentiate. They may yield all of the pressure symptoms and even pulsation in aneurismal areas. The following points assist in differentiating them:—(a). *Knowledge of a primary focus of malignant growth.* (b). *Absence of the auscultatory signs of aneurism.* (c). *Rapid emaciation.* (d). *Absence of expansile pul-*

History clear.

Fluoroscopy.

Error unlikely.

Confusing condition.

Offer difficulties.

Important points.

*In the author's experience, however, this has been of a remittent type, and relieved by absolute rest, at least in the early stage of the disease.

†Of these cases 50% have already died suddenly, in several instances without developing the more marked symptoms.

sation either upon ordinary inspection or more definitely as determined by fluoroscopic methods. In two cases seen recently by the author a certain amount of aneurismal dilatation *co-existed* with a large mediastinal growth, in the one case a sarcoma, in the other, carcinoma. Certain

Aneurism may co-exist.

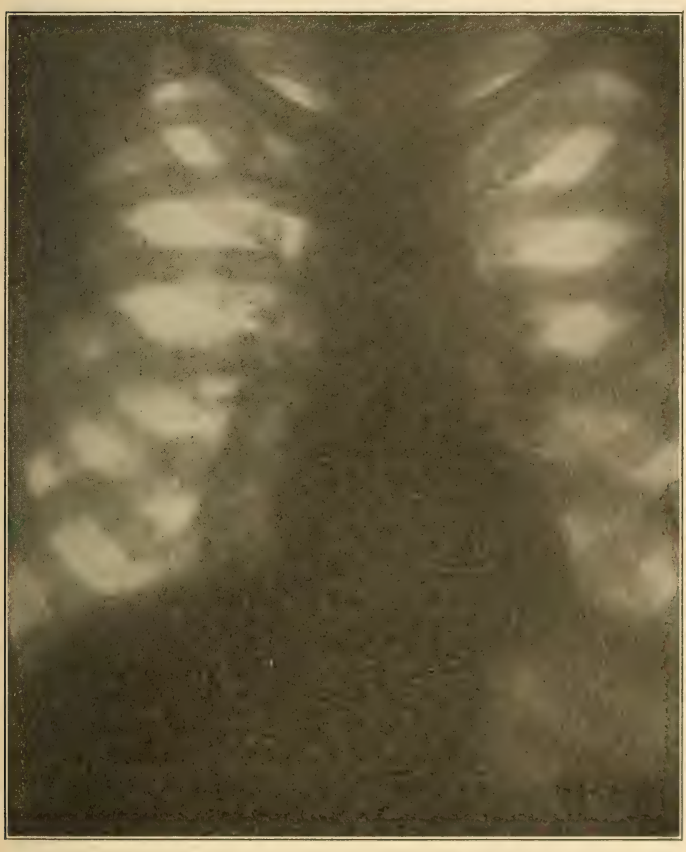


Fig. 113.—Malignant growth producing stenosis at the cardia and extending upward in the mediastinum. Heart and dilated aorta are represented by the deeper shadow. Confirmed by repeated photography and finally by autopsy.

vascular growths with definite expansile pulsation may appear and force the diagnostician to rely wholly upon auscultatory signs. In such instances occasional errors in diagnosis, or failure in positive determination are unavoidable.

Excusable error.

Final Considerations and Conclusions.—*Certain cases of aneurism yield no symptoms save those determined by X-Ray examination. Here as elsewhere, a knowledge of the normal heart sounds is of the first impor-*

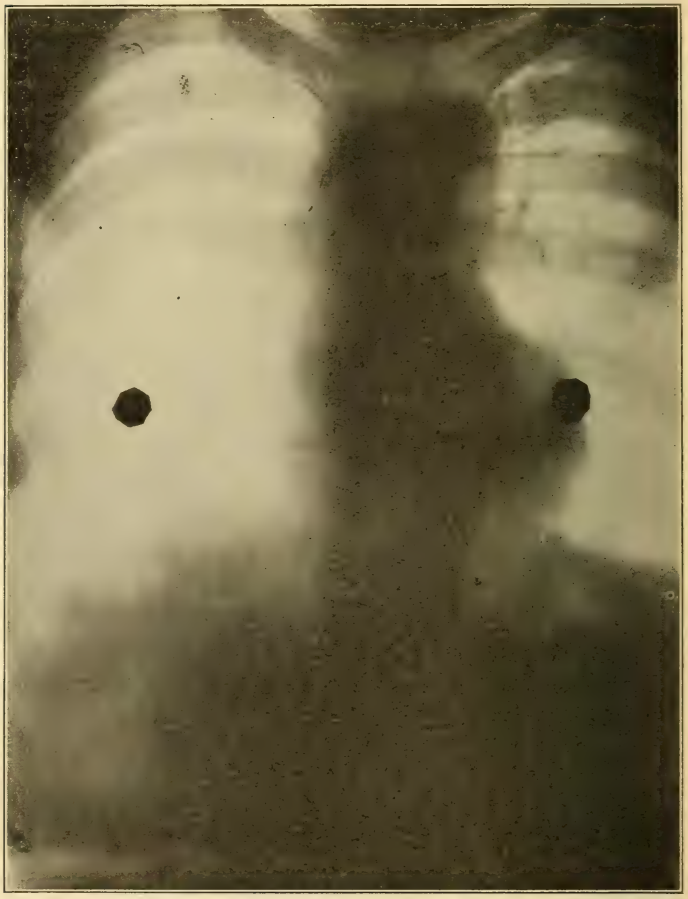


Fig. 114.—Malignant obstruction at cardia, growth occupying mediastinum.

tance. All ringing, and distinctly metallic second sounds at the base should be regarded as suspicious when their points of maximum intensity are found at some distance from the proper valvular area. Tan-

gential inspection should never be omitted and in doubtful cases yielding percussion dulness the effect of absolute rest should be determined. In all walking cases of aneurism any existing area of dulness is perceptibly reduced by a few days of rest, and the *daily variability of pressure symptoms* as related to the occupation or the pursuits of the individual should be considered. *The temperature record, strength and nutrition* should be watched; *unilateral loss of knee jerks is suggestive and the history of past syphilis* or external signs of a denied lesion are most important. It should be remembered that *senile pericarditis is often aneurismal,* and that *active pressure ulceration* of the pulmonary structure *may produce physical signs of tuberculosis,* and finally, that with the fluoroscope one may usually see and accurately measure the sac, determine true expansile pulsation and accurately record the changes induced by rest or treatment.

All important data.

ENDOCARDITIS.—Definition.—An inflammation of the endocardium, acute, subacute or chronic, benign or malignant, affecting chiefly the opposed surfaces of the valves, but more rarely the mural endocardium, and tending to produce transient lesions or permanent vegetations or deformities more or less seriously affecting valvular action.

Varieties*.—In general there are three classes: (1). *Verrucose,* in which the valves are the seat of vegetations, finely papillary in appearance, yellowish white in color and composed of spindle cells derived from the fibrous tissue or endothelium, leucocytes, fibrin threads and granular material. They are often *capped* by layers of fibrin. (2). *The ulcerative form,* characterized by predominance and rapid extension of a necrotic process present to a slight degree in the preceding form, the clinical symptoms being largely those of septic intoxication. (3). The *sclerotic* or fibrous form characterized by its slow development, its chronicity and its tendency to deform valvular structures. *It is impossible to clearly separate the acute benign from the acute ulcerative form, save on clinical grounds, and a chronic endocarditis of any type may follow an acute attack.*

Three types.

Etiology.—No specific micro-organism can be charged with the responsibility for acute endocarditis. The pneumonococcus, staphylococcus, streptococcus, and more rarely the colon bacillus and the bacilli of influenza, tuberculosis and typhoid have been found in

*G. A. Gibson aptly says: "It is impossible to draw any definite line separating these lesions from one another. They form a chain of which the ends, though extremely diverse, are yet united by links presenting a perfect gradation."

endocardial lesions. *Acute rheumatism, the acute forms of septic infection, and the toxines of various acute infectious diseases* are associated with both the benign and ulcerative cases. The sclerotic forms are caused by the same factors as are active in arterio-sclerosis generally.

Frequency.

It is probable that an endocarditis exists in nearly every severe case of acute rheumatism (its most common cause) and that many spontaneous recoveries from transient unrecognized lesions occur. Children are especially liable to this acute complication of rheumatism, 60–80% presenting demonstrable lesions. In adults the percentage is probably about 5c%, though a lower figure is usually given by authorities.

Age.

Associated ailments.

Chorea and acute tonsillitis are both closely associated with this affection. though in choreic cases many murmurs are present which may prove ultimately to be functional or accidental. Any of the exanthemata and especially scarlatina and diphtheria may be complicated by it and gonorrhœa in the adult is sometimes associated with the ulcerative form.

Emboli.

The peculiar structure of the vegetations in verrucose endocarditis makes it possible for fragments to be swept away by the blood stream and produce emboli in the brain, lungs or other viscera, thus accounting for some of the sudden deaths and paralytic seizures, encountered in chronic heart disease.

Subacute Endocarditis.—This form presents few symptoms, other than a murmur, and fever may pass unnoticed unless it appear in an afebrile interval of the primary disease. Palpitation and inconstant symptoms of dyspnœa, or *more often* a *sense of precordial oppression* are usually present though often overlooked.

Precordial oppression.

All cases of acute infections or chronic diseases such as gout, diabetes and Bright's disease should be carefully watched; in the former the heart should be examined at each visit, for oftentimes serious valvular lesions are overlooked and the patient is allowed to be up and about under conditions full of danger and making certain the establishment of a chronic lesion. **Ulcerative Endocarditis.**—The symptoms are essentially those of (a). The typhoid state. (b). Of pyemia, and are usually definite though latent cases occur.

Offers many difficulties.

Chill, high fever and sweats associated with valvular murmur are the more characteristic features but it may closely simulate typhoid fever, malaria and meningitis. Petechiæ are usually present, multiple abscess formation is common, splenic tumefaction, often sudden from infarct, may occur, gastro-intestinal symptoms may be predominant, and, rarely, a septic arthritis develops. *The diagnosis depends largely upon the presence, or the development of a valvular lesion with septic and usually embolic manifestations.*

Chronic Endocarditis.—This permits a division under two distinct classes. (1). Cases following rheumatism or other acute infections. (2). Those due to sclerotic or fibroid changes resulting from alcoholism, syphilis, gout, chronic nephritis or mere senile changes.

The resulting valvular alterations may cause the most extreme deformity or be comparatively slight and the symptoms are merely those discussed under the head of valvular diseases.

PERICARDITIS.—*The etiologic factors* in pericarditis are identical with those of endocarditis discussed in the preceding section, 50% of the cases being attributable to rheumatism and representing chiefly the younger ages; Bright's disease and tuberculosis being active factors particularly at older ages. Traumatism and diseases of malnutrition play a somewhat larger part and the acute infections generally a lesser one than in endocarditis. Indeed it is a not uncommon terminal event in many chronic diseases. About 50% of the cases are dry or plastic and males are chiefly affected. It is a frequent accompaniment of cases showing valvular lesions or may be coincident with an acute or subacute endocarditis.

Morbid Anatomy.—The changes are identical with those of pleurisy both as to the nature and course of the inflammatory changes and the variable constitution of the exudate, hence we have not only dry pericarditis and pericarditis with effusion, but the latter may be serous (the common form), hemorrhagic, purulent or gaseous.

Symptoms.—Like the pleura the pericardium forms a potential cavity, its layers being reflected upon the heart and upwards upon the great vessels for an inch or more and its parietal layers structurally continuous with and adherent to the diaphragm below and the cervical fascia above. In cases of effusion it is converted into an actual receptacle capable of holding from one to three pints or even larger quantities in exceptionally chronic cases; thus it may form within the mediastinum a tumor so large as to cause many of the *pressure symptoms* fully discussed under aneurism, page 205, and furthermore seriously embarrasses the action of the heart itself and the adjacent pulmonary structures. Its close relation to the pleura makes its participation in a pleurisy common, as also the involvement of the pleura in a pericarditis. Having these facts in mind its symptoms suggest themselves. They are (a) *pain*, often severe, (b) *fever*, (c) *accelerated pulse*, (d) *dyspnœa*, (e) *precordial oppression*, (f) *friction sounds*, (g) *signs of effusion*.

Anatomic relations important.

Pressure.

Extension of process.

Symptoms.

Marked pain is present in about three-fourths of the cases but is variable in its site and intensity, being felt usually chiefly over the

Cardinal symptoms.

base of the heart, in the epigastrium or even referred to the left arm and shoulder as in angina pectoris. *The fever* is usually moderate and of variable duration, lasting from a few days to several weeks according to the course of the inflammatory process and is of a septic type if suppuration occurs. *The pulse* may not only be accelerated but markedly arrhythmic. *The dyspnœa* is often marked but not excessive unless effusion occurs when it may even reach orthopnœa. Slight *cyanosis* may be present in the early stages and is marked in the presence of large effusions. *Precordial oppression* may be present even in the early stage and is intensified in large effusions. *The friction*

Chief sign.

sounds may be single, double or triple, are usually heard chiefly at the base and to the left of the median line and are *superficial, rubbing, creaking, shuffling or grating sounds* though at times assuming the qualities of a harsh valvular murmur or a crepitation. *They are seldom as*

Characteristics.

directly related to the heart sounds as endocardial murmurs, are likely to shift their points of maximum intensity and change their quality from day to day and are often associated with marked local tenderness and increased audibility under stethoscopic pressure. The double sound is most common, the second pulmonary sound is accentuated, murmurs have no definite transmission as in endocardial disease, and, friction fremitus is often present. If *effusion* occurs it usually *relieves* the *pain*

Effusion.

and *is manifested by changes in the cardiac outline* varying with the extent of effusion but tending to assume a pear-shaped or triangular form

Dulness.

with the base downward. *The dulness is more marked than in ordinary dilatation, the apex beat often appears higher than normal, is within the left border of dulness and may entirely disappear in large effusions. The acute angle ordinarily formed by the relative dulness of the right*

Rotch's sign.

heart and the liver boundary is rendered obtuse (Rotch's sign), *the heart sounds are diminished* over the area of dulness *and pressure signs* become prominent as the effusion advances. An *irritative cough* sometimes of brazen quality may be present, compression of the left lung may give rise to *dulness under the left scapular angle with harsh or tubu-*

Ewart's sign.

lar breathing (*Ewart's sign*), *cyanosis* deepens, *dyspnœa* increases and the patient is usually distressed, depressed and apprehensive. The symptoms of pressure affecting mediastinal structures (page 205), should be noted, the pulse may be dicrotic or paradoxic, and respiratory venous collapse (see p. 176) may suggest extensive inflammatory adhesions. Occasionally but not always the lesser effusions show marked changes in the level of dulness with changes in posture.

Chronic Adhesive Pericarditis.—This common sequel offers great

diagnostic difficulties. Occasionally as in pleurisies it is suggested by the persistence of modified friction over the heart. If extensive the diaphragm may be so dragged upon by adhesions as to show the one pathognomonic sign, viz., *systolic recession of the lower left thoracic zone or of the interspaces representing the diaphragmatic attachment.* So also there may be persistent systolic filling and diastolic collapse of the jugular veins. In persistent adhesion there is a dilatation and hypertrophy of the hampered heart much less favorable as regards prognosis than that of endocardial disease.

Suggestive signs.

Definite symptom.

Heart and veins.

Diagnosis.—Aside from the factors mentioned the diagnosis of effusion may require the use of an aspirating needle of small calibre which is most safely introduced into the crevice formed by the ensiform and the rib margin on the right side, the needle being directed towards the point of the right shoulder. *This procedure is not to be undertaken unless prompted by real necessity.* The long persistence of an effusion and the occurrence of a septic temperature point to *pyo-pericardium* and the substitution of marked resonance (of the form of the sac), for the dulness points to *pneumo-pericardium*, an excessively rare condition. While theoretically and indeed usually the diagnosis of this condition is simple, in practice, cases of the utmost difficulty may be encountered and more than one highly skilled clinician has introduced the needle into a *dilated heart* usually, fortunately, without serious result. It is necessary, therefore, to bear in mind the peculiar featurs of *pericardial* effusion dulness, its more marked character, its greater upward extent, its peculiar form and its extension beyond the apex beat if that be visible. Massive long standing effusions have been mistaken for left sided pleurisies. *Hydro-pericardium* occurs as a part of general dropsy and is seldom mistaken for actual inflammatory effusion because of the associated conditions and the usual absence of primary fever.*

Aspiration.

Pus.

Gas.

Error possible.

Important features.

Transudate.

MYOCARDITIS.—The diseases of the myocardium may be divided into acute and chronic, the former occur chiefly in connection with the severer forms of infectious diseases such as scarlatina, typhoid, typhus, malignant endocarditis and septic intoxications in general and constitute one of the most common and serious complications of diphtheria, and the heart may show enormous dilatation, almost abso-

Acute.

Associated ailments.

* In a case observed by the author some years ago an aneurism of the aortic arch occupying the right lower chest and associated with a markedly dilated and displaced heart closely simulated in the character and extent of dulness a marked pericardial effusion; fortunately the aneurismal signs were sufficiently distinct to prevent any error, though friction sounds of cardiac rhythm were present at the base.

lute inefficiency, or indeed produce death instantly or with slight warning.

Diagnosis clear.

Symptoms of Acute Myocarditis.—Marked arrhythmia, palpitation, vertigo and weak, rapid or excessively slow pulse, dyspnœa, cyanosis and diminished urine may be associated with a clear mind or in some cases delirium. The heart sounds are weak, fœtal and lacking in normal accentuation and there is usually no difficulty in making out by percussion the outline proving dilatation.

Chronic myocarditis may accompany:—(a) *coronary sclerosis, embolism or thromboses*, (b) *chronic valvular disease or senile degeneration producing the condition known as brown atrophy*, (c) *fatty overgrowth and fatty degeneration.* Fatty overgrowth lacks the actual degenerative changes of the latter form though marked fatty infiltration may impair the function and nutrition of the muscle and may even affect the papillary muscle and endocardium. In both cases the heart is soft, yellow and enlarged. In actual fatty degeneration the muscle fibres themselves show granules and oil droplets and disappearance of nuclei and normal markings; both conditions may be associated with primary anæmias, obesity, a cachetic state and senile atrophy. True degeneration may also accompany prolonged sepsis or severe infections, chronic hypertrophy, dilatation, pericarditis, coronary disease or various chronic toxæmias.

Suggestive symptoms.

Symptoms.—*Differential* ante mortem diagnosis of fatty heart is seldom possible though the age of the patient, the habits, or the presence of obesity may be suggestive. On the other hand there are many suggestive symptoms as applying to chronic myocarditis in general. *In the arterio-sclerotic cases* sudden attacks associated with *pain*, varying from a dull ache to extreme angina, may be associated with *increased arterial tension*, no doubt due to vaso-motor spasm. Such conditions are frequently transitory and the pain may be substituted by *marked precordial oppression or sense of suffocation.* In the more severe or terminal cases *pulmonary edema is marked, cyanosis* extreme and the patient dies suddenly or lapses into unconsciousness. *Edema or general anasarca may be present but is often lacking.* In all varieties the *pulse may be markedly arrhythmic, unequal, and variable as to tension, and the heart sounds show abnormal accentuation and usually a fœtal quality. The first sound at the apex is frequently absent or greatly diminished* and under treatment a murmur may appear and the sound be partially re-established. *Systolic murmurs*, most commonly of the mitral and aortic orifice, are frequently observed and *bradycardia or tachycardia*

Variability. Pulse. First sound. Murmurs.

may be present or may alternate in the same patient. *In the terminal stages of the disease one of the most characteristic features is the failure of response to such drugs as digitalis and oftentimes indeed to any* Digitalis.

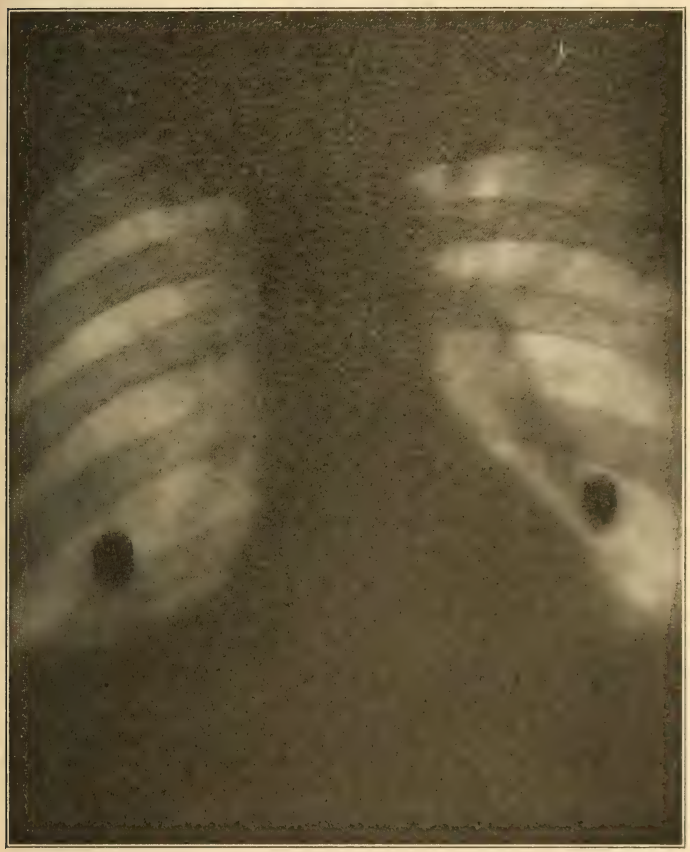


Fig. 115.—Flabby heart, masked chronic myocarditis with atypical angina pectoris. Death from cardiac asthenia a few weeks later. Note obtuse angle formed by right border of heart and upper border of liver, simulating Rotch's sign of pericardial exudate. No fluid in pericardium.

drug or to rest, In the earlier stages however the same therapeutic measures, aside from the administration of digitalis, as are successful in ordinary valvular lesions may succeed here. **The terminal symptoms**

are merely those of *extreme cardiac incompensation*, frequently associated with *angina pectoris* or other evidences of vaso-motor spasm; attacks of *severe dyspnœa*, sometimes coming on during sleep, *orthopnœa, and if sudden death does not intervene the gradual development of general anasarca. An enormous number of these cases are overlooked and fail to receive the early treatment which might save much suffering and prolong life and usefulness.* As before stated *the vital error* consists in the failure to demand the normal heart sounds in any case, too great stress being laid upon murmurs as indicating heart disease. So also actual dilatation may entirely escape observation.* *A great number of these cases are encountered in connection with chronic alcoholism and indeed in those who are far from being either occasional or habitual drunkards.*

Safeguard against error.

RUPTURE OF THE HEART.—*This condition occurs only in persons having a degenerated heart muscle* and in such may result from a fall, strain or other traumatism, such as is apparently insufficient to produce such an accident. Usually it occurs in the anterior wall of the left ventricle, rarely in the right and causes death instantly or within a few moments, preceded by severe pain, oppression and symptoms of collapse. This condition is medico-legally important.

A clinical curiosity.

ANEURISM OF THE HEART.—This rare condition may be saccular and associated with acute or chronic endocarditis or myocarditis, or due to pericardial adhesion or gummata. As a clinical curiosity dissecting aneurism may be encountered. Death occurs usually from rupture and the disease cannot be diagnosticated ante mortem.

FOREIGN BODIES IN THE HEART.—The organ is much more tolerant than is generally supposed; exploring needles, sewing needles, hat pins and even a knife or bullet may not cause death, indeed a small needle may be introduced with a minimum risk of a fatal result and it has even been suggested or actually employed as a therapeutic *dernier ressort* for cardiac stimulation. According to W. G. Thompson, war records show an astonishing *exemption* of the heart from bullet wounds though the statement is based upon the Civil war records which antedated the use of high penetration projectiles.

Remarkable tolerance.

NEW GROWTHS OF THE HEART.—New growths, tubercu-

* In a case observed not long since an old patient went to his death in an extremely high altitude upon the basis of expert opinion, though to the author's knowledge he had had severe recurrent incompensation with marked transient dilatation, due to myocarditis, for several years.

losis, syphilis, various forms of sarcoma and carcinoma, and more rarely the myomata, lymphomata or fibromata may involve the heart. It may be the seat of abscess in pyemia, malignant endocarditis and acute myocarditis, or the seat of degenerative or cysticercus cysts.

SITUS VISCERUM INVERSUS.—As a part of the general transposition of organs occasionally observed as a congenital phenomenon we may find the heart upon the right side, the left ventricle being anterior and the apex in the usual relation to the nipple as in its normal position. Such transposition is sometimes complete as regards the other organs, sometimes partial. Apparent transposition of the heart is common but usually proves to be due to a combination of unilateral pressure and lack of support.* As a temporary condition extreme degrees of displacement may be associated with pleural effusions.

Extremely rare.

ARTERIO-SCLEROSIS.—Definition.—A chronic degenerative change involving especially the arterioles on the one hand and the larger blood vessels on the other, the two conditions being invariably associated.

Morbid Anatomy.—The arterioles are the seat of arterio-capillary fibrosis, irrespective of the order of the degenerative changes, the endothelial and sub-endothelial tissues, the elastic layer and the media being involved, with resulting loss of elasticity, a lessening of the calibre and increased peripheral resistance. In the larger arteries there is a cellular infiltration about the vasa vasorum, involving the subintimal and medial layers, which is followed by degeneration and necrotic changes tending to weaken the arterial walls. Should an infiltration nodule undergo fatty and hyaline degeneration an *atheromatous abscess* is formed which may be converted into an ulcer by necrosis of the endothelial wall. When infiltrated by calcareous matter the nodules form the well known *atheromatous plaques*. This process may extend so far as to constitute a *senile atheroma* with its pipe-stem vessels, rigid and tortuous in outline, as seen in very aged persons or the victims of *premature* senility. *Phlebosclerosis* is less common but may be marked. The aorta is the most frequent site of atheromatous changes and an arterio-sclerotic process in the cerebral arteries is the most frequent cause of apoplexy, usually because of aneurismal dilatation.

*In a case seen with Dr. B. J. Merrill, apparent complete displacement was due to almost complete tuberculous excavation of the right lung, with enormous hypertrophy of the left.

Occupation. Habits. Heredity. Sex. Age.

Etiology.—Again one finds that Venus, Bacchus, Vulcan, Mammon, Mars or Minerva, may be held responsible, though such conditions may result from simple ageing forming one of the natural terminations of advanced life, and, on the other hand, may be distinctly hereditary, *arterio-sclerosis being one of the most strikingly hereditary of all known conditions.* Men are affected far more frequently than women and more or less change in the blood vessels must be *expected* in men who have reached the meridian of life and in many instances are seen in young men and even in children. *These latter constitute cases of premature or precocious senility* so far as the blood vessels are concerned.

Symptoms.—*Inspection.* The superficial vessels are prominent (the temporals especially so), and oftentimes show a pulsation resembling that seen in aortic regurgitation but more *vermiform.* In marked cases the apex beat of the heart is displaced downward and to the left and the heaving beat of hypertrophy may be present.

Usual conditions.

Palpation.—The finger recognizes readily the thickened artery of varying rigidity, which may be rolled under the finger as a distinct tube when the artery is shut off above and the nodular or plaque like irregularities may be noted when present.

Important sign.

Auscultation.—The aortic second sound is sharply accentuated, and, as a rule, a systolic murmur in the aortic area simulating aortic stenosis but due to the roughened intima may be present. (See also endocarditis.)

General Symptoms.—Many curious transitory attacks may be experienced, the chief of which is vertigo, though transitory spasmodic seizures, monoplegias and hæmiplegias may also occur. Apoplexy is of course always to be feared and if the coronary arteries are involved attacks of angina pectoris and intermittent and irregular pulse are most troublesome. As a general condition arterio-sclerosis leads to apoplexy, degeneration of the coronary vessels and myocarditis, and, to interstitial nephritis. The symptoms are so distinctly those of associated or dependent lesions as to need no extended description. In its atheromatous form it is the essential factor in the production of aneurism.

Predisposing causes.

ANGINA PECTORIS (dolor cordis, heart pang).—For purposes of convenience this complex is given a place by itself. *It is nearly always a symptom* of arterio-sclerosis, though marked degrees of arterial degeneration may exist and this symptom be absent. Persons subjected to severe mental strain and worry, who have reached or are nearing the meridian of life, are especially subject to attacks and men

who work with their hands may be comparatively free from it even though the arterial changes are marked. Attacks may be precipitated by violent emotion, such as fear, anger, grief, and, not infrequently, by sudden physical strain.* When patients are subject to attacks very slight influences may be adequate causes. So also after over-eating, indigestion, through fermentative changes and distension of the stomach may cause an attack of angina or close the earthly career of a victim of arterio-sclerosis or myocarditis. Sudden deaths of this kind are frequent in the experience of every coroner.

Excitants.

Acute gastric dilatation.

Symptoms.—*The essential symptoms are an intense pain in the region of the heart, and a sense of impending death. In typical cases the patient is rigid, apprehensive, pallid and perhaps perspiring, and such may be afraid to speak or breathe while the attack persists.* It is usually of short duration and is often characteristically relieved by such a drug as amyl nitrite. *All these symptoms may, however, be greatly modified even in true angina, and the term cannot well be limited to the severer cases.* Instances in which there is but moderate oppression may be encountered and the pain varies greatly in location, transmission and severity. It is usually associated with marked oppression; in extreme cases this may be described as crushing or vice like, shooting or aching; extension to the left shoulder and arm is common, and it may even be localized in the *right* upper chest though this is less frequently the case.

Variable in severity, and seat.

Associated Lesions.—Though arterio-sclerosis is the chief cause angina often accompanies regurgitant lesions of the aortic valve, mitral stenosis, acute aortitis, chronic myocarditis and aneurism. *In its milder forms it may hardly be differentiated from a pseudo-angina encountered in victims of hysteria, neurasthenia and certain forms of dyspepsia or attacks related to the excessive use of alcohol and tobacco or sexual excess.*

Various causes.

DISEASES OF THE ABDOMINAL ORGANS.

THE ABDOMEN (*Abdo.*, I conceal).—Excepting the brain this is the most abscure region of the human body because of the shifting, air containing, obscurant intestinal coils, its inaccessibility to direct examination, and, the comparatively slight value of certain of the physical methods so valuable in thoracic diseases. On the other hand the

Obscure region.

*In a case recently observed a series of attacks terminating in cerebral embolus and death followed a street car accident involving a sudden stop which threw the patient backward with such force as to snap the holding strap held in his left hand.

Recent advance.

recent development of the laboratory side of the subject has been remarkable both as to extent and practical value. At the present time, while one must admit that positive diagnosis is often impossible, it is equally true that, too often, recourse is had to diagnosis by surgical exploration without any serious preliminary attempt to exhaust the more modern methods of purely medical diagnosis.

Boundaries.

Divisions.

Contents.

Topography and Regional Divisions.—The spine, the lower costal margin, the iliac crest, Poupart's ligament and the pubic symphysis form the superficial boundaries of the abdomen and its cavity is roofed by the diaphragm which shelters and imparts respiratory movement to the liver, spleen, and, to a less degree, the kidneys and stomach. The upper limit of the *cavity* is indicated by a transverse line at the level of the base of the ensiform, the lower by the true pelvic brim. A *vertical* intersecting a *horizontal* line at the navel divides the abdomen into *four quadrants*. *The upper right* contains the liver, gall bladder, head of pancreas, kidney, the hepatic flexure, a portion of the transverse colon and the pylorus. *The left upper*, the left extremity of the liver, the spleen, splenic flexure, a part of the transverse colon, left kidney, the fundus of the stomach and most of the pancreas. *The right lower* contains the ascending colon, cæcum and the vermiform appendix, the left lower the descending colon and the sigmoid flexure. The uterus and ovaries are anatomically *pelvic* and not abdominal and the lesser intestinal coils occupy the whole lower area.

Landmarks.

The anterior superior spine, iliac crest, pubic spine, lower ribs, costal margin and *the ensiform* are the chief bony landmarks. The *linea alba, the navel* and the *rectus muscles*, divided into segments by the lineæ transversæ and bounded externally by the lineæ semilunares, are easily distinguishable in the abdomen of thin persons. The *umbilicus* usually lies from an inch and a half to two inches above a line connecting the anterior superior iliac spines and is opposite the tip of the 3rd lumbar spine, and, the *10th and 12th ribs* and *the projection representing the 9th cartilage* are also important landmarks. *The abdominal aorta* bifurcates opposite the 4th lumbar vertebra three-fourths of an inch to the left of and below the navel, and *the ilium and jejunum* occupy chiefly the centre and lower portion of the cavity, the ilium largely on the left and jejunum on the right and below. *The colon* is less shifting and movable and ascends from the cæcum upwards to the right, then (hepatic flexure) transversely, crossing above the umbilicus (transverse colon) under the left costal margin and (splenic flexure) downwards, inwards (descending colon and sigmoid flexure) to term

inate in the rectum. The hepatic flexure lies under the arch of the liver, the splenic even higher under the left ribs. The transverse colon, sigmoid flexure and cæcum are relatively superficial and the *vermiform appendix* lies with its base at a point representing the intersection of the outer edge of the right rectus with a line drawn from the anterior superior iliac spine to the umbilicus (*McBurney's point*). It lies behind and to the inner side of the cæcum with its apex downward, outward, inward or rarely upward.

THE LIVER AND GALL-BLADDER.—The normal percussion outline of the liver is shown by the following table.*

		Middle Line.	Mammillary Line.	Midaxillary Line.	Scapular Line.
Upper limit.	Deep dulness.		Fourth space.	Seventh space.	Ninth space.
	Superficial dulness.	Blends with heart dulness	Sixth rib.	Eighth rib.	Tenth rib.
Lower limit.		Hand's breadth below base of xiphoid.	Costal margin or somewhat above or below it.	Tenth space	Blends with kidney dulness.

It should also be noted that the left lobe extends nearly to the left nipple, the interlobar notch lying in the median line. In children the liver lies lower, in old persons sometimes higher and in others lower than the position given.

The gall-bladder lies just within the projection of the 9th costal cartilage near the outer edge of the right rectus.

The spleen lies in the left upper quadrant just outside the spine in close relation to the diaphragm above, the left kidney, posteriorly, and the colon, stomach and small intestines below. It is oval, flattened and in length from three to five inches and it lies parallel to and within the area of the 9th, 10th and 11th ribs, its anterior margin reaching a line dropped from the sterno-clavicular joint to the point of the 11th rib. In two-thirds of its surface it is parietal.

The Kidneys.—These lie in apposition to the posterior wall, being in contact above with the liver on the right side and the spleen on the left and move with respiration. The right is slightly lower than the left

* Hutchinson and Rainy. "Clinical Methods."

and each lies two-thirds within the mid-clavicular line, the lower border of the right being one inch, that of the left 1½ inches above the navel. Posteriorly, they lie within a parallelogram represented by two verticals drawn respectively one inch and two and three-fourths inches from the spinous process, and two horizontals representing the 11th dorsal and 3rd lumbar spines; thus about one-third of their surface is covered by the 11th and 12th ribs and their lower extremity lies from an inch to an inch and a half above the crest of the ilium.

TECHNIQUE OF ABDOMINAL EXAMINATIONS.—All varieties or subdivisions of clinical methods are applicable to this region. *The first step* when time permits consists in the *administration of a cathartic*, a procedure, however, by no means always employed nor indeed necessary, for its chief purpose is to remove masses of fecal matter which might be mistaken for abdominal growths and in some instances it has the positive disadvantage of producing gaseous distension and prolonged peristaltic activity which may obscure the findings.

Removal of fecal masses.

The attitude of the patient is most important. The *head and shoulders, particularly the former*, should be well raised, the feet firmly placed, and the patient himself be in a secure, easy and unconstrained, dorsal posture. To secure muscular relaxation of the walls it is usually recommended that the knees should be drawn up, but often, *better relaxation may be had with the legs extended or but slightly flexed.* The breathing should be easy and natural with the mouth open and the patient's attention should be diverted by conversation and inquiry relating to other phases of the case. The palpating hands should be warm, the light should be adequate and its source behind the patient, and no clothing or other covering embarrass the examiner. The *flat* of the palm and fingers, *not the tips, should be used and tender areas should not be immediately approached;* nor does the discovery of one justify repeated and purposeless punching and poking. *In palpating movable organs or growths respiratory movement should be met directly by the tips of the fingers* at right angles to the free margin, the hand being firmly but gradually depressed in deep expiration and allowed to rise laggingly and shift slightly upwards to meet the descending border in inspiration. *If the hand moves the organ or growth under examination the opposite palpating hand should be almost motionless as the other brings the organ or tumor to it.* After tolerance is established the finger tips may be used much more freely for the determination of consistence and outline. If *excessive pain or tenderness* be encountered one should note whether it is superficial and felt on light pressure, or only on deep

Relaxation

Diverting attention.

Use of hands.

Important data.

pressure and whether it is distinctly localized or diffuse. *Malingering or hysterical patients* can often be misled or diverted by conversation or by attracting their attention to some special region which is being explored by the one hand, while the supposed tender point is palpated by the other. *In some cases nothing less than general anæsthesia suffices.* The dorsal posture is the best for general purposes but the lateral is often of value in the palpation of the spleen or kidneys or for the purpose of "spilling" the intestinal contents of a large bellied person to render more accessible the lateral region under examination. (A special procedure (dipping) is referred to under ascites, page 12.) In many instances the patient may with advantage be placed upon the hands and knees or in the knee chest position to bring a growth forward against a thin and relaxed abdominal wall, the procedure being seldom successful otherwise save under anæsthesia. Finally, *all portions of the abdomen should be thoroughly examined not forgetting the hernial openings.*

Useful device.

Varied postures.

THE PHYSICAL EXAMINATION AND ITS SPECIAL APPLICATION.—Points to be Determined.—(a). *The condition of the organ under observation* (size, outline, condition of surface, mobility, tenderness). (b). *The presence of swelling or tumors* (intra- or extra-abdominal, size, outline, surface, mobility, tenderness, associated symptoms). (c). *Presence of pain or tenderness* (true or false, intensity and character, exact location, superficial or deep and the effect of pressure). Pain increased on pressure ordinarily suggests inflammation.

General Inspection.—*The nutrition, musculature and abdominal outline are first noted. General distension* may be due to tympanites, ascites, chronic tuberculous peritonitis, *great* dilatation of the stomach or colon, pregnancy or ovarian tumor. *Localized distension or swelling* to hysteria (phantom tumor), ventral hernia, abscess, intestinal obstruction, new growths, dilatation or displacement of the stomach.

Outline.

General retraction may indicate extreme emaciation, carcinoma of the stomach and especially of the cardia, lead poisoning, meningitis, chronic diarrhœa or dysentery. *The skin* may show an eruption of the acute exanthemata, syphilis or skin diseases, areas of pigmentation atrophy and the various hues and discolorations already discussed under the "outward signs of disease," or the white or bluish striæ indicating present or past distension by tumor, pregnancy, ascites or excessive fat; *the linea nigra* or dark median line may indicate a pregnancy, the *superficial veins may be enlarged* in the case of portal or vena caval obstruction, and point to hepatic cirrhosis, tumors or

Surface.

Veins.

prolonged ascites in the former, or the pressure of abdominal growths or thrombosis in the latter. *If the superior cava be obstructed* the direction of the blood flow in the large superficial veins (readily determined by emptying one and watching its refilling) will be downward, in other conditions it is upward. *If the visible distension is most marked in the pubic surface-segment, obstruction below the liver is suggested.* Aortic obstruction as indicated by enlargement and prominence of the epigastric and internal mammary arteries is a clinical curiosity.*

How increased.

Visible Peristalsis.—In the presence of emaciation and a thin and relaxed abdominal wall peristaltic movements may be noticeable and are readily made more pronounced by snapping the surface with the finger nail or wet towel, splashing upon it a little ether or applying the faradic current. *Normally the movements are deliberate and wave-like and occur usually without borborygmi,* but in acute obstruction they may seem to be fiercely attacking the stenosed region. Normal

Variability.

stomach peristalsis runs from left to right; that of the transverse colon from right to left, a point slightly diminished in value because of the rare reversed peristalsis in marked pyloric obstruction. In relation to obstruction of the bowel, acute or chronic, the sign is of distinct value,

"Ladder pattern."

one of its commonest forms, the "ladder pattern" peristalsis, running from the ilio-cæcal valve region towards mid-abdomen.† *In sigmoid obstruction* there is increased violent peristalsis along the course of

"Cooing."

the colon and a cooing or loud rumbling associated with the disappearance of distension which at times sharply localizes the point of constriction. *One of the commonest causes of increased visible peristalsis is stenotic dilatation of the stomach.*

THE LIVER.—Inspection sometimes shows a lower border shadow descending with deep inspiration. *The normal percussion* outlines have already been given, (see page 225). *Auscultation* is of no value save in the detection of peritoneal friction in inflammatory lesions or abscess and the most useful physical method is *palpation.*

Technique.

Mobility.

Palpation.—The normal liver can often be palpated easily if the wall be relaxed and the liver edge *met* by the fingers rising *with* the wall in deep inspiration. *Directness and immediateness of movement are characteristic of the liver* as compared with that of the mova-

* See congenital heart lesions.

† In two such instances observed by the author marked obstruction due to appendicitis existed for a period of 30 years, the diameter of the tube having been reduced to that of a crow quill. In one case the recurrent attacks had been so violent as to have led to an operation for gall stones, the pain being to some extent referred to the hepatic region.

ble kidney or a pyloric growth and is absent only when adhesions exist or there is decided limitation or complete absence of diaphragmatic descent. Any decided *resistance* in the liver edge as palpated indicates a morbid change in the hepatic tissue, such as cirrhosis, chronic congestion or tumors. Rarely, and more often in children, *the liver pulsates directly and expansively* from tricuspid regurgitation and, yet more often, there appears a *transmitted pulsation* due to a dilated right ventricle.

Fixation.

Consistence.

Pulsation.

Percussion.—As regards percussion it should be said that *a reduced area is especially significant if it involves the region of the left lobe* as this is earliest affected in atrophic cirrhosis. *Diminution of the upper* (thoracic) *area* may arise from shrinkage of the liver but is more commonly only apparent and due to emphysema of the lung or, rarely, diaphragmatic hernia. This area varies with deep inspiration and expiration not only by reason of the descent of the organ but because of the descent of the lung border. *Auscultatory percussion* will be found valuable particularly in connection with the left lobe or in deciding whether a tumor is hepatic in origin or attachment. The stethoscope should be placed over the thoracic area of *superficial* hepatic dulness while the finger taps the surface in radiating lines until the decided change of note indicates that hepatic tissue has been passed. Attached tumors will usually yield the hepatic note under these conditions. One of the *commonest errors* of the tyro in the examination of the liver or spleen is that of beginning palpation too high, great enlargement of either organs being frequently overlooked.

Valuable method.

Conclusions.—*Upward extension* of dulness in *the hepatic area* suggests enlargement of the liver, pleural effusion or adhesions, pulmonary consolidation, hepatic or subphrenic abscess. *Diminution of upper boundaries* indicates hepatic atrophy, asthma, emphysema, *pneumothorax*, gaseous distension of a *subphrenic* abscess, hepatic ptosis and rarely, massive pericardial effusion or cardiac dilatation. *General enlargement* may be due to passive congestion of cardiac origin, the commonest cause, hypertrophic cirrhosis, leukæmia, fatty liver, amyloid disease, *syphilis*, cancer, gumma, abscess, pyemia. *General shrinkage* suggests atrophic cirrhosis, the commonest cause, with acute yellow atrophy as a clinical curiosity.

Modified percussion areas

Irregularities in Outline.—Such may be due to congenital defects, various enlargements and displacements, atrophic cirrhosis and indeed practically all of the conditions noted in a preceding paragraph. Of special significance are the nodular irregularities (often showing crateriform depressions) associated with secondary carcinoma, and

tertiary syphilis. *Glissonian cirrhosis* may produce marked shrinkage and deformity whereas the tiny granular elevations of *atrophic cirrhosis* can be palpated only under exceptional conditions, as when the wall is thin and lax, though *the sharp resistant edge in an advanced lesion is striking. Fluctuating swellings or tumors* modifying the outline are usually due to a distended gall-bladder, abscess or hydatid cyst; the last yielding a peculiarly exquisite thrill, echoing the percussion stroke. *It should be remembered that an enlarged liver, easily palpable, with a smooth hard surface, suggests amyloid disease, passive congestion and fatty liver. Roughening suggests cirrhosis or tuberculosis; nodulation, carcinoma and syphilis.* Finally it will be noted that mere increase or diminution in the hepatic area yields no information as to the actual size of the organ unless checked carefully by an examination of the contiguous structures.

Nodules.

Hydatid thrill.

Caution.

THE GALL-BLADDER.—Palpable only when distended it may then form a pear-shaped tumor,* smooth and elastic, anchored above but freely movable from side to side, and in the absence of adhesions, rising and falling in respiratory rhythm *with the liver itself.* Rarely, a palpable crepitation may be obtained if gall stones are present or it may be nodular from malignant disease. In long standing cholecystitis it may undergo atrophy.

Mobility.

Crepitation rare.

THE SPLEEN.—A greatly enlarged spleen and more rarely one scarcely more than palpable may be seen to move with the respiration; furthermore, in any case of splenomegaly, a marked prominence of at least the left upper quadrant is produced.† As in the case of the liver, *auscultation* may reveal friction sounds in the presence of perisplenitis, or the organ may be anchored by adhesion, but *it is ordinarily freely and directly movable with respiration, and palpation is the only method yielding important results.* The position of the patient should be right lateral if minor enlargements are to be noted, as in typhoid fever or other acute infections, and the right hand should make pressure posteriorly while the left makes palpation. Abdominal distension defeats palpation save in great enlargement and *the normal spleen is not palpable.*

Mobility.

Proper attitude.

* These tumors in rare instances attain an enormous size, Albuan Doran having recently reported one simulating *ovarian cyst.* (British Medical Journal, June 17, 1905.)

† In a case recently shown by the author to the Minnesota State Society a leukæmic spleen filled the entire abdomen, actually reaching the right anterior superior spine of the ilium, and strange to say the patient showed no impairment of health, change in color, or malnutrition and declined to cease work or take treatment.

If greatly enlarged the dorsal position is to be preferred and the condition can hardly be overlooked, unless with a tense wall the careless or hurried examiner fails to get below the actual border or to distinguish between muscular resistance on the one side and the splenic mass on the other. In these cases the *chief notch* in the anterior border is sharply defined and quite distinctive. Students at first *palpate too deeply* to recognize minor enlargement. *In all cases where doubt arises as to the nature of a large tumor in the splenic area it should be remembered that a splenic growth or tumor is superficial, that its dulness is marked and can be carried directly back to its normal area behind the 9th, 10th and 11th ribs, that it ordinarily moves directly with respiration and that if the colon be inflated splenic tumor dulness is not impaired.* On the contrary, large *renal tumors* extend more deeply, are relatively fixed or immovable, are likely to be reniform or nodular and are crossed by an area of resonance if the colon be distended with air. As in the case of the liver a downward displacement of the diaphragm may produce an apparent increase in the lower splenic area just as an emphysematous lung or a pneumothorax may cause an apparent diminution in its upper percussion area.

Source of error.

Important data.

Low diaphragm.

Causes of Enlargement.—*Excessive chronic enlargement* is most frequently due to leukæmia, splenic anæmia or chronic malaria; lesser degrees may be met with in cirrhosis of the liver, rickets, pernicious anæmia, passive congestion, or actual portal obstruction. *Acute enlargement* is most often encountered in septicæmia, malarial fever, typhoid and typhus, erysipelas, acute miliary tuberculosis, tuberculous peritonitis, cerebro-spinal meningitis, smallpox, diphtheria, scarlet fever, relapsing fever, infarct, plague and certain other tropical diseases.

Enlargement of the spleen is nearly always uniform, abscess being an occasional, and hydatids or carcinoma an excessively rare cause of irregular outline.

KIDNEYS.—The normal kidney yields *no* results to inspection or auscultation and *indefinite* ones to percussion. When enlarged by disease it may be evident in the anterior lumbar region and be readily accessible to percussion and palpation and, bulging may also be manifest posteriorly. The sarcomata in children, and cystic, tuberculous or hydronephrotic kidneys in the adult, are the most frequent causes of enlargement.

Palpation.—The dorsal and *semidorsal* postures are the best, and, in the latter, relaxation is assisted by supporting the shoulders and buttocks and bringing the hand and arm of the side under examination

Posture.

Technique. obliquely downward and forward across the abdomen. One hand of the examiner makes pressure over the floating ribs or just below them posteriorly, while the other is pressed deeply downward and slightly upward outside the rectus border on a level with the umbilicus. If *the kidney is movable* it will slip downward in deep inspiration so as to be grasped between the tips of the fingers of the two hands. *The normal kidney yields to palpation only its lower border and this only under favorable conditions. Any greater degree of mobility constitutes movable kidney.* If the organ is so movable and displaceable as to suggest a mesonephron it is a *floating kidney* and such are sometimes overlooked because at the moment they occupy some distant portion of the abdomen, being frequently found low down in the region of the pelvic brim or displaced across the median line.*

Degrees of mobility.

Source of error.

Percussion.—The semidorsal position is best adapted for percussion which is usually superfluous, uncertain and often misleading. Occasionally displacement is suggested by the substitution of tympany for normal dulness or an unusual extension of the latter may point to diseased conditions.

Characteristic Features.—The reniform shape and peculiar consistence of the kidney are readily recognized unless the organ be markedly inflamed or the seat of a new growth. Under these conditions *the tumor is readily palpable, relatively or absolutely fixed, regular or irregular, smooth or nodulated, according to the nature of the morbid process. If the organ be merely movable as is so often the case, its maximum displacement occurs near the end of full inspiration,* a fact readily explained by its relation to the diaphragm and the directly movable spleen and liver. *Furthermore, such displaced kidneys are replaceable into the flank or normal kidney position and in fixed renal tumors inflation of the colon establishes a band of resonance crossing or wholly obscuring the percussion dulness anteriorly. Secondary abscess in the renal region is not uncommon* as a sequence of neglected appendiceal abscess, empyema, subdiaphragmatic abscess, caries of the vertebra, etc., and one must consider the *globular elastic or possibly fluctuating tumors* of hydro- or pyonephrosis, the *irregular cystic tumors*, the *denser more nodular malignant growths* and the *bogginess or induration* of the perinephritic suppurations which *tend posteriorly* rather than anteriorly,

Peculiar form.

Lagging.

*It is sometimes advised that examination should be made with the patient leaning forward in the standing posture with the elbows or hands supported on a chair or table. The author has never found this procedure of special value except with extraordinarily lax abdomens or when the kidney was greatly enlarged.

in contradistinction to tumors of the kidney itself. Chemic and microscopic examination of the urine, careful consideration of primary factors and the use of the aspirating needle assist diagnosis. Valuable aids.

THE STOMACH.—General Considerations.—Despite modern advance there is a remarkable lack of positive knowledge in regard to the capacity, dimensions and normal position of the human stomach, and, indeed, of the vagaries of its secretory activities. The stomach of the newborn baby has a capacity of but one or one and a half ounces, yet in from six to eight years this is increased to one quart and the capacity of the adult stomach varies from one to two quarts. *Five-sixths of the stomach lies to the left of the median line;* the transverse measurement varies from twelve to sixteen inches; the depth from above downward is but six inches and *the distance between the pyloric and cardiac orifice but one and a half inches. The fundus lies in the left cup of the diaphragm* and the upper limit of percussion resonance normally extends to the fifth left interspace in the parasternal and midclavicular lines, while the lower runs from one to two inches above the navel. *The pylorus should lie midway between the right sternal and parasternal lines at the level of the xiphoid, the cardiac orifice lying about an inch and a half to the left, opposite the* 11*th dorsal vertebrum.* Absolute lines cannot be drawn for the delimination of an organ so distensible and freely movable. In general it may be said that a stomach whose lower border reaches the navel is abnormal, either in size or position.

Lack of data. Capacity. Position and dimensions Fundus. Cardia and pylorus.

PHYSICAL EXAMINATION.—Inspection.—This yields valuable results more often than is generally supposed, and, especially in cases with thin, relaxed, abdominal walls, the outline of a distended stomach may be clearly defined. Growths, especially of the pylorus, are often seen to move with respiration and normal (left to right) or very rarely reversed (right to left) peristalsis may be present, often affording valuable corroborative evidence of pyloric obstruction, the gastric waves being large and deliberate and taking from ¼ to 1 minute to pass across the field. In the congenital pyloric obstructions of children the tumor is often readily seen.

Valuable. Outline. Growths. Peristalsis.

Fluoroscopy and Transillumination.—By giving the patient massive doses of bismuth subnitrate (20 gms.) mixed with porridge or some similar food, fluoroscopic or skiagraphic evidence may be obtained, while occasionally Einhorn's method of transillumination may greatly assist inspection. The *gastrodiaphane* is merely a moderately rigid bougie or soft tube carrying an electric light. The patient drinks two glasses or more of water, after which the instrument is introduced

Bismuth. Gastrodiaphany.

Normal. and the electric circuit completed. In a normal stomach an illuminated zone appears in the region of the left lower segment of the normal fundus and extends about to the lower margin of the left lung. It is triangular in form, the apex being downward and slightly above the Pathologic. umbilicus. It naturally follows that in gastroptosis or dilated stomach (gastrectasia) the illuminated area is lower and larger. The information gained through inflation of the stomach is usually so much greater that the method is seldom required.

Palpation.—This is chiefly used to determine three points: (1). *The presence of a tumor.* (2). *Localized tenderness.* (3). *Succussion. The palpable tumors of the stomach most often involve the pylorus and lie to the right of the median line between the margin of the ribs above, and the navel below.* Areas of thickening, often plaque like in feel may Old ulcers. indicate an old ulcer base and though adhesions frequently occur, anchoring the stomach to adjacent organs and greatly complicating and obscuring diagnosis, ordinarily such growths move freely and indeed oftentimes are so widely removed from the usual site as to suggest growths of distant organs. *Their movement and position in connection with inflation then becomes of especial importance.* In very thin flabby abdomens one must be on his guard against mistaking other structures, perhaps normal, for gastric growths. *Intermittent stomach rigidity* over the fundus may be encountered in cases of pyloric stenosis and through relaxed thin walls or in cases of enteroptosis the pylorus may be directly palpated and felt to harden and relax with an associated "squirting" sensation (*spritz geräusch*) and, if a soft tube be used the point may Useful method. be distinctly felt as it is coaxed along the lower border to the pylorus. *Succussion* is obtained by placing one hand over the lower left ribs posteriorly while making sharp dipping movements anteriorly. Any splashing sound suggests dilatation or gastroptosis *if it occurs more than 7 hours after a meal.* The value of this sign has been greatly Overestimated sign. overestimated and is practically limited to its occurrence before breakfast or on a fasting stomach. *In general it merely indicates slight motor insufficiency*, or, if it be obtained within narrower limits but *by very gentle tapping it strongly suggests gastric atony.** Finally, *dilatation of the stomach may, though rarely, occur without superficial splashing and the sign is undoubtedly in some degree dependent upon the condition of the abdominal wall itself.*

Percussion.—Ordinary percussion is of little use and need not be discussed. It was formerly the custom to introduce a pint or more of

* Sahli distinguishes between "deep splashing" and "superficial splashing."

water and percuss the dull area produced, but both the boundary and the muscle tone are better established by inflation.

Auscultatory percussion is distinctly useful, particularly if preceded by inflation. Normal gastric tympany is represented by the segment of a circle, being limited above by hepatic dulness, but includes Traube's space and has a curved border below which *should not reach the navel.* Thus contraction of the left lobe of the liver, shrinkage of the left lung and marked distension of the stomach tend to enlarge it. By careful auscultatory percussion the upper right and left limits may be somewhat extended, though the change of note due to the lung and left hepatic lobe will be distinctly marked.

Best method.

Areas.

Technique.—*The bell of the stethoscope is placed just below and to the left of the ensiform cartilage and percussion* is carried from it in radiating lines, in accordance with the boundaries outlined above, either from the stethoscope outward until the note changes, or, from the surrounding negative areas towards that of gastric resonance. It is well to shift the position of the stethoscope and repeat the percussion in order to check results. So also in the case of a growth, if the stethoscope be placed over it and a similar method employed, one may often determine its connection with the stomach. An elaborate description of the tones is unnecessary as the veriest tyro can distinguish the *changed note* that indicates that the stomach border has been passed.*

Auscultation.—This almost valueless method finds its only use in the detection of esophageal "swallowing sounds," the succussion sound already referred to, and the sizzling or fizzing of active fermentation and the test for hour glass (bilocular) stomach.

Slight value.

Turck's Gyromele.—This interesting instrument lends itself to inspection, palpation and auscultation and consists of a flexible piece of steel such as a piano wire inclosed within a rubber tube and bearing a soft sponge. Being introduced into the stomach it is attached to a little hand instrument which causes rapid revolutions and will distinctly show the sponge as it passes along the lower gastric border. Its inventor uses it not only for diagnostic but for therapeutic purposes.

Often useful.

Inflation of the Stomach.—*Methods.* (1). The oldest and simplest involves the administration of one dram of sodium bicarbonate in a wine glass full of water, *followed* promptly by an equal amount of tartaric acid similarly dissolved. *Theoretically* it is open to the objection that

Old method.

*The note is uniform in quality and pitch so long as the *stomach* is percussed, though intensity depends upon the distance of the stroke from the stethoscope.

serious danger may result, especially in ulcer, from overdistension; *practically* this danger is slight yet the method is decidedly inferior to the following one. (2). After introducing the stomach tube, air is pumped into the stomach with a Davidson's syringe or if the patient be tolerant and the physician courageous the inflation may be accomplished by blowing into the tube. As the degree of inflation is wholly under control in this matter the procedure has almost wholly superceded the older one. The distension should not be great and the same contra-indications govern it as in the use of the stomach tube itself.*

Best method.

Caution.

Inflation brings into prominence tumors of the anterior wall and obscures those of the posterior wall. In general, gastric growths are cylindrical at the pylorus, broad and flat on the fundus.

Tumors.

Interpretation of Outline.—The normal boundaries have already been given; if abnormal, one should carefully note whether there is merely *a normal or approximately normal area in an abnormal position* (gastroptosis), whether there is *both abnormal position and increased area* (gastroptosis with ectasia), or finally, whether there is *general enlargement without marked displacement* (simple dilatation).

Ectasia and ptosis.

THE STOMACH TUBE AND ITS USES.—One should use a soft rubber tube carrying ample fenestrations at its extremity and a large terminal perforation. It should be surmounted by a glass funnel and some physicians introduce, a few inches below, a glass tube as an indicator. According to the author's experience the simpler the stomach tube the better are the results and the elaborate and complicated special tubes are unnecessary.

Simple requirements.

Contraindications.—Under certain conditions the introduction of the tube is unjustifiable and dangerous, and it is always necessary to balance the value of the result to be obtained and the possible danger experienced.† The following conditions *usually* forbid the use of the tube *in those not habituated to its use.* (a). *Extreme weakness and exhaustion from whatever cause.* (b). *Advanced myocarditis.* (c). *Recent hæmatemesis or tarry stools.* (d). *Advanced arterio-sclerosis* or *past cerebral hemorrhage.* (e). *Pregnancy.* (f). *Aortic aneurism.* (g). *Terminal pulmonary tuberculosis* especially if hæmoptysis has occurred. (h). *High grades of emphysema.* Furthermore, in elderly persons of apoplectic build and tendency the first passage of the tube usually involves

* Coincident distension of the colon by injections of water is employed by some clinicians.

† That this is not imaginary was shown not long since in a case observed by the author in which a large *soft* tube passed directly through the base of a *carcinomatous* ulcer.

an amount of straining and congestion that is extremely dangerous. *These restrictions need only apply to hemorrhagic cases and aneurism in those habituated to the use of the tube.*

Technique of Introduction.—The patient should be reassured and told to obey orders absolutely, swallowing when told to do so and breathing deeply through the nose. *Sharp commands* are often successful and effective in shutting off nervous gagging or retching or attempts to pull out the tube, *which should be warmed* by placing it in hot water, *held like a pen and introduced promptly and firmly* without haste and without regard to temporary obstruction. The patient may be asked to swallow as it reaches the pharynx and there need be little fear that it will enter the larynx; if it does, the fact is at once evident, the tube is removed and no harm done. *No considerable force is at any time necessary nor should it be employed.* The depth to which it should normally be passed is from 16 to 22 inches from the teeth and the average distance is usually indicated upon the tube. In ectasia it will need to be more deeply introduced and the fact is diagnostically suggestive. The patient's head should be slightly forward, the mouth open, *the tongue in*, and *neither in the introduction of a stomach tube nor in that of an esophageal bougie is there the slightest reason for introducing the finger into the patient's mouth*, a procedure which increases gagging and distress and often involves a bitten digit. *To obtain the contents*, the patient is asked to take a deep breath and strain as at stool, while leaning sharply forward. This movement may be assisted by firm pressure over the region of the navel made either by the patient or the operator. In some cases it may be necessary to attach a syringe bulb or Politzer's bag to the tube and make suction.* From time to time a tube may be blocked and the bulb may be used to force in jets of air or, the obstruction may disappear if the tube be slightly withdrawn and reintroduced; this latter procedure being often necessary in any event. *The first attempts* to introduce a stomach tube are usually associated with more or less violent retching and straining and are greatly dreaded by the patients, but they may be assured that after three or four introductions the discomfort will be slight.

Important suggestions.

Keep fingers out of mouth.

Expression of contents.

Tube blocking.

Habituation rapid.

EXAMINATION OF GASTRIC CONTENTS.—Aside from the methods described under examination of the abdomen, our knowledge of gastric ailments depends upon a consideration of the case history in

*Such a bulb can readily be fitted with a glass tube that accurately fills the calibre of the stomach tube and the device is better than the use of a compression bulb on the tube itself.

Physiology.

connection with a chemical examination of the stomach contents taken after a test meal. After ingestion of any ordinary articles of food there follows a period of rising acidity. The hydrochloric acid combines promptly with the proteids and alkaline bases leaving no free acids to inhibit fermentation by bacteria, hence for from 15 to 40 minutes after a meal one would find hydrochloric acid abundant in combination, but absent as a free acid, while starch transformation would progress unhindered and lactic acid would appear because of fermentation or introduction with the food. A second period follows during which the affinities of the proteids are satisfied, *free* hydrochloric acid appears and fermentation ceases. *After certain test meals, this period should be attained an hour after the meal is ingested and one thus has a definite normal by which* abnormal variations may be measured. This second period *is followed in two hours or more* by a third characterized by a *fall* in the acidity. *An excess of proteids requires much acid for combination and means in all cases a delay in the appearance of free hydrochloric acid; an excess of carbohydrates acts in the opposite way*, their acid affinities being promptly satisfied.

Application.

Effect of diet.

Points to be Determined.—Primarily one seeks for *free or combined hydrochloric acid*, the latter representing a loose chemical combination with alkali-albumin and undergoing progressive transformation by means of an acid and the action of the digestive ferments which results in the production of (1). *Acid albumin.* (2). *Proto- or hetero-albumoses.* (3). *Deutero-albumoses and finally, peptones.*

Certain Standard Test Meals are here given but it should be remembered that any meal will give results if its constitution be known and that finical exactitude is not essential.

Ewald's Test Breakfast.—One or two slices of stale bread or one or two baker's rolls and from 300 to 400 c.c. (8–12 oz.) of weak tea or water. This represents 35–70 gms. (1 to 2 oz.) of solid, but the *exact* amount is not material. It should be taken in the morning and retained for not less than one or more than one and a half hours.

Boas' Test Breakfast.—One tablespoonful of oatmeal is added to one quart of water and boiled down to one pint. It should be taken plain save for a little salt and removed in not less than one nor more than one and a half hours. *This breakfast is especially valuable in cases of suspected cancer of the stomach as it introduces no lactic acid, the determination of which is so important in this disease. In such cases the stomach should be washed out the night before or in the morning an hour before the meal is taken;* in fact *Ewald's* breakfast seldom causes

any confusion, lactic acid not being introduced in sufficient quantity to produce the ordinary clinical reaction suggesting carcinoma.

The Test Dinner of Von Leube and Riegel.—400 c.c. (12–14 oz.) of soup; 50 gms. (2 oz.) (or two ordinary slices) of wheat bread; 100 to 200 gms. (3–6 oz.) finely divided beefsteak and 200 c.c. (6 oz.) of water constitute the meal. It is removed about 4 hours later.

The Macroscopic Appearance of the Stomach Contents.—One hour after an Ewald test *breakfast* the appearance should be that of thin porridge, the bread in particles rather than distinct fragments and little or no mucus present. The filtrate should be clear or thin yellow and of slight odor. *Anacidity* or marked *hypoacidity* is indicated by unchanged or slightly modified bread fragments, slow filtration and a musty odor. *Hyperacidity* by greater fluidity, foamy surface, stale, acid odor and rapid deposit of sediment. In *catarrhal gastritis* the thick slimy content with evidence of subacidity is characteristic and in hyperchlorhydria with *excessive motility*, the stomach may be entirely empty.

Normal.

Abnormal.

Other constituents are discussed under "vomitus," p 249, and other headings.

QUALITATIVE TESTS.—The following procedure is simple and convenient:—(a). *Test filtrate with congo-red paper or solution* (congo-red 1, distilled water 100), or add 1 or 2 drops of an alcoholic solution of di-amido-azo-benzol (.5%); free acid is indicated by blue discoloration with the former, or red with the latter reagent.*

Simple tests for HCl.

(b). *In the absence of HCl, lactic acid* should be sought by placing in the test-tube 2 *drops of carbolic acid*, 6 *drops of neutral tincture of ferric chloride and adding water until a deep but clear amethyst color appears. The filtrate is then added drop by drop and if lactic acid be present a clear thin yellow reaction appears;* no attention need be paid to doubtful reactions. Lactic acid is always abnormal if appearing one hour or more after a test breakfast.† (c). *Acetic and butyric acids* are revealed by the odor of vinegar and rancid butter produced upon heating. Butyric acid appears as small oily drops of characteristic

Lactic acid.

When abnormal.

Other fermentation acids.

* Or, a few drops of resorcin solution (resorcin 5., cane sugar 3., alcohol 100.), or, phloroglucin solution (phloroglucin 2., vanillin 1., alcohol 100.) may be mixed with an equal amount of filtrate in a porcelain capsule or on any white surface and evaporated to dryness, *a beautiful marginal rose color indicating free HCl.*

† The reaction is intensified by shaking up the filtrate with ten times its volume of ether, evaporating, adding a few drops of water and applying test.

odor if a bit of calcium chloride be added to an ethereal residue.* These *volatile fatty acids* redden litmus paper held over the tube during boiling and *indicate fermentation.* Lactic acid is the only fermentation acid of marked clinical importance; strongly suggesting carcinoma and proving fermentation and the absence of the HCl which normally checks fermentation.

Relative importance.

Pepsin and Pepsinogen.—*With normal or increased hydrochloric acid no test is necessary* and in any event pepsinogen may be disregarded.

Test.—Prepare three tubes. (1). *Water* 10 c.c.+*pepsin* 5 *grs.*+HCl *dil.* 3 *drops.* (2). *Filtrate* 10 *c.c.* (3). *Filtrate* 10 *c.c.*+*HCl dil.* 3 *drops,* add to each a disc of egg albumin about 1.5 mm. thick, and 10 mm. in diameter.† Tube (1) serves as a standard of comparison. Digestion in No. 2 indicates both pepsin and HCl; in Number 3. Number 2 *failing,* it shows the presence of pepsin or pepsinogen alone. Digestion should be complete in about three hours at blood heat but considerable variation is allowable.‡ As a rule pepsin activity and acidity run parallel, but this rule may be departed from in extreme hyperacidity and certain cases of subacidity, so also, if motor sufficiency be present, even a total absence of acid and ferments may cause few symptoms.

Simple tests.

Relation to HCl.

Rennin (lab ferment).—*Test.* Add to 10 c.c. of milk 3–5 drops of gastric contents, keeping mixture at blood heat. The appearance of coagulation within 15 minutes indicates rennin. It may be entirely absent in carcinoma, achylia gastrica and atrophic gastritis, but is the most resistant of the ferments.

Most resistant.

Lipase.—The elaborate test reaction for lipase is of interest only to laboratory workers as its only clinical importance lies in the diminution or entire absence of this ferment in achylia gastrica or atrophic gastritis and this may be assumed if both HCl and the ferments are absent. In any event it is but slightly active.

Slight value.

Albumin.—This should be absent one hour after a test breakfast.

Starch.—The digestive transformation of starch begins in the mouth where ptyalin rapidly converts it into *amidulin.* This becomes *erythro-*

Transformations.

*A more exact test for acetic acid demands treatment with ether, evaporation, neutralization of the ethereal residue with dilute sodium hydrate and the addition of a drop or two of dilute ferric perchloride, but the results are not worth the labor.

† These should be constantly on hand and are readily prepared, from the *lightly* boiled egg, preserved in glycerine and washed before using.

‡The methods ot Thomas and Weber depending upon the digesting of a solution of casein in 0.2% of hydrochloric acid and the still more complicated one of Mett is too cumbersome for the general practitioner.

dextrine which strikes a violet or mahogany brown color with Lugol's solution. This in turn becomes *achroödextrine* (unaffected by iodine) and maltose (readily detected by Fehling's solution). It is evident that if the gastric secretion be normal the hydrochloric acid will prevent more than a partial conversion of the starch, despite the rapid action of the ptyalin, the extent of which depends upon the thoroughness of mastication and the character of the food as swallowed in relation to the penetration of the acid encountered. It is checked when the proteid affinities are satisfied and free acid appears. Tests.

Clinical Inference.—*A marked starch reaction suggests hyperacidity, a marked achroodextrine reaction, diminished hydrochloric acid and a marked Fehling's reaction decided subacidity or absent* HCl.

Testing the Motor Power of the Stomach.*—15 grs. of salol should show excretion by the kidney in from 1 to 1¼ hours and be entirely excreted in 27 hours. It appears as salicyluric acid which strikes a violet color with ferric chloride; most conveniently applied by placing a single drop of the latter upon the wetted test paper. The salol is not affected by the gastric juice. Salol test.

Second test. An extremely simple test consists in allowing a patient to eat cranberries, blackberries or other stony fruits at night, removing the contents in the morning when the residue show the seeds even if no actual stenosis is present. Rough test.

Better probably than either method is the removal of the stomach contents in the morning, a full test dinner or ordinary meal having been taken the night before. Under these conditions and also two hours after the Ewald test meal a stomach should be either entirely or approximately empty. Best method.

In actual stenosis, benign or malignant, solid food remnant representing the residue of several previous meals may be present. Stasis.

The *absorptive power* of the stomach may be tested by the administration of 10 grains of potassium iodide, in capsules, which should produce in the sputum the characteristic iodine reaction with starch paper within 10 or 15 minutes. Iodide test

QUANTITATIVE TESTS for Free, Total, and Combined Hydrochloric Acid.—*Take three portions* (10 *c.c. each*) of the *unfiltered* gastric contents; add to one 3 or 4 drops of phenolphthalein solution (1% alcoholic solution). *This substance being the indicator for organic acids, acid salts and both free and combined HCl, upon titration with an alkaline solution of known composition, will measure the total acidity.* Total acidity.

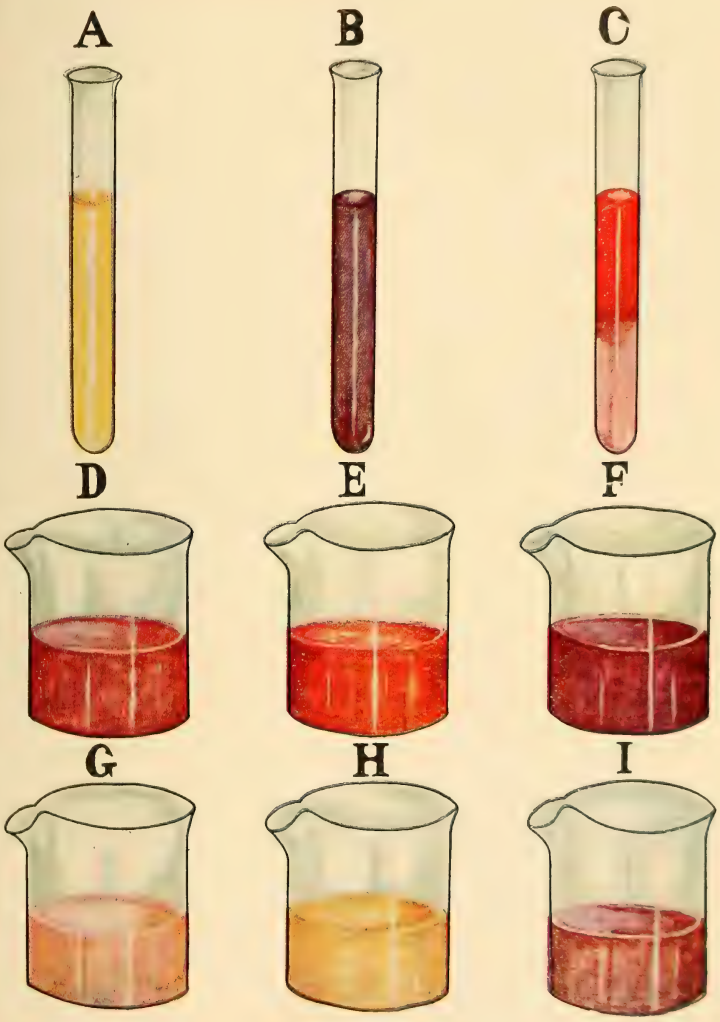
To the *second* portion add 3 or 4 drops of alizarine

*The more elaborate and exact tests of Mathieu, Sahli, Goldschmidt et al. have not been included.

DESCRIPTION OF PLATE I.

A. *Lactic Acid* reaction.
B. Test fluid before adding stomach contents in *lactic acid* test.
C. Di-amido-azo-benzol reaction for *free HCl.* (qualitative test).
D. True terminal reaction for *total acidity* (Phenolphthalein).
G. Antecedent color change in same test, *not* the terminal reaction.
E. Color of stomach contents after adding test solution for *free HCl.*
H. Terminal color reaction some test. (Color also nearly represents that assumed by stomach contents after adding test fluid (alizarin sol) for combined HCl).
F. Terminal color reaction *combined HCl.*
I. Antecedent color, *not* true end reaction same test. Test really represents all acids in stomach contents except combined HCl (see text), the result obtained being subtracted from the previously determined total acidity.

PLATE I.



Important Color Reactions in Connection with the Examination of the Gastric Contents. (Drawn from Actual Specimens.)

solution (1% aqueous sol. alizarin monosulphonate of sodium). *This being the indicator for free acids and acid salts, measures the degree of acidity except for the combined HCl* (physiologically active acid loosely combined with the proteids); *hence by subtracting the reading thus obtained from the total acidity* (which *does* include the combined acids) *one gets the amount of the combined acids.* To the third portion add three or four drops of dimethyl-amido-azo-benzol (0.5% alcoholic sol.); *this being the indicator for free hydrochloric acid.*

Combined HCl.

Free HCl.

Sol. 1 *is cloudy, white or grayish. Sol.* 2 *a bright yellow. Sol.* 3 *a cherry red if free acid be present.* The three solutions are now each to be titrated with decinormal sodium hydrate solution,* one c.c. of which will neutralize 0.00365 gms. of HCl.† *When neutralization is reached and just passed the true end color reactions appear.*

Color of mixtures.

End Reactions.—These are:—*In solution* 1 (for total acidity) *a distinct and persistent red* which does not deepen upon the addition of more NaOH. In solution 2 (for free acids and acid salts) a *pure violet color;* in solution 3 (for free HCl) a *fixed yellow.* One must carefully note at the commencement of each titration the exact reading indicating the height of the solution in the burette and again take it on the first appearance of the end reaction. *The results may be expressed in exact terms by multiplying the amount of acid known to be neutralized by* 1 *c.c. of the NaOH sol.* (.00365 gm.) *by the amount of that solution used and this again by the number of c.c. of filtrate tested. Example.* Assuming that 6 c.c. are required to neutralize the 10 c.c. of stomach contents, and knowing that each NaOH unit neutralizes 0.00365 of acid, the formula becomes:—6 x 0.00365=.02190 gms. (for 10 c.c.) x 10=.219%. This is commonly and more conveniently expressed by figures based upon the assumption that as 10 c.c. of stomach contents required 6 of NaOH for neutralization 100 would require 60, or if 8 were used 80, and so on. Using the latter form of notation we find that normal free HCl varies from 20 to 40, i.e., .073 to .146%. Total acidity from 40 to 60, i.e., .146 to .22% and combined acidity about 25, i.e., .09%, one hour after the test breakfast of Ewald or Boas. After the Riegel meal (3 hours) the total acidity is about 110, free acid 44.

Red (total acidity). Violet (comb. HCl). Yellow (free HCl).

Actual and percentage figures.

Usual notation.

Normals.

* A decinormal solution=$\frac{1}{10}$ of the molecular weight of the substance, in gms., to 1000 c.c. of distilled water, hence in the case of NaOH the molecular weight being 40 the solution contains 40 parts to 1000 of distilled water. (A normal solution=the molecular weight of the substance in 100 parts of distilled water.)

† The fact that the three right hand figures correspond to the number of days in the ordinary year is an allowable crutch for a limping memory.

The process is extremely simple and no more complicated methods are at all essential for *clinical* work. It *should be remembered that if free HCl is present it is the only acid in combination.*

Recapitulation.—Sol. No. 1 (phenolphthalein) reacts to *free acid, acid salts, and the loosely combined acids,* is *grayish white* and cloudy and gives a *red* terminal reaction. *Sol. No.* 2 (alizarin) *measures the same acid contents as No.* 1 *less the loosely combined acid,* thus necessitating for an estimate of the combined acid the subtraction of the result obtained, from the figures representing the total acidity. The *original color* of No. 2 is yellow, the *end reaction violet. Sol. No* 3 (dimethyl-amido-azo-benzol) *reacts only to free acid, is originally cherry red, and its end reaction is a brilliant yellow.* Finally, the *normal total acidity is* 40–60 (.146 *to* .22%); *the combined acidity* 25 (.09%); the *normal free HCl* 20–40 (0.73 to .146%) after a test breakfast.

Negligible.

Sources of Error.—Töpfer's method as here given is by far the most accurate and convenient for clinical work and while slightly inaccurate when free hydrochloric acid is absent especially in the presence of an excessive amount of the organic acids, these errors are not sufficient to disqualify the test for the general practitioner.*

Effects of Diet.—Variations in acidity, particularly in free acid, will of course depend somewhat upon diet and an excessive amount of saliva *may neutralize the gastric contents;* in such cases the addition of a few drops of dilute HCl and 2 or 3 drops of ferric chloride solution to the stomach contents causes a red color reaction. As the acid affinities of starchy foods are readily satisfied and the demands of proteid substance much greater, *the highest acid values are found in meat eaters and the lowest in vegetarians.* The more liberal the diet and the better the nourishment the more the acid. It is evident also that the real value of our figures is represented by the "combined" and "free" acid percentages.

Rough deductions.

The Diagnostic Bearing of the Foregoing Methods.—For hasty work the mere determination of free HCl is most readily achieved by the use of congo-red paper easily procured of any chemist though it tells us nothing save the bare fact that HCl is present. This being the case however the presence of normal ferments is usually a fair presumption. Wood warns the physician against placing too much dependence upon the first test meal, rightly stating that, owing to the emotional disturbance, the results may be misleading. The same rule may be

* The methods of Sahli, Reissner, Martius and Luttke may be found in works devoted to clinical diagnosis, but are too complicated for the use of the practising physician.

applied to several successive tests, if, as seldom happens, the nervous disturbance continues to be excessive. Ordinarily we have to consider four conditions. Variable findings.

(1). **NORMAL ACIDITY** (euchlorhydria). This excludes achylia gastrica, chronic gastritis and usually carcinoma of the stomach. It does not exclude gastric atony and dilatation, nervous dyspepsia or the rare cases in which carcinoma develops upon an old ulcer base.*

(2). **HYPERCHLORHYDRIA** (0.2% +).—We distinguish between the secretion of excessively *acid* gastric juice during digestion (hyperchlorhydria proper) and the same condition appearing in the fasting stomach and representing more or less continuous *hypersecretion* (*gastrosuccorrhœa* (a) continuous—*Teichmann's disease,* or (b) periodic or intermittent—*Rossbach's disease*). As regards *hyperchlorhydria proper,* its chief clinical importance depends upon its frequent association with ulcer of the stomach and the *possibility* that it is a factor in the production of that lesion. Simple hypersecretion moreover is common in neurotics without ulcer symptoms, in the gastric crises of tabes dorsalis, biliary engorgement, and, as a temporary condition due to exhaustion, emotional disturbance, constipation, dietetic errors, overuse of alcohol and tobacco or even to special articles of food. Ulcer. Various associations.

Symptoms.—Its symptoms are no doubt chiefly due to an associated hyperæsthesia of the mucosa. The most characteristic feature is the combination of slight or decided digestive disturbance (epigastric oppression, pyrosis, regurgitation, nausea, perhaps vomiting and occasionally *diffuse* pain and headache) *with a clean tongue.* The attacks appear from one to three hours after a meal, are relieved if vomiting occurs, or often (temporarily) by taking nitrogenous food and are in all cases ameliorated by the administration of an alkali. Slight *diffuse* tenderness may be present and constipation is the rule. The stomach contents are usually clear, acrid to the taste, of low specific gravity and will of course show high acid values† and unconverted starches. The urine at the height of an attack is usually neutral and deposits phosphates. In acute attacks the pain may be severe and strongly suggests ulcer save that it is more diffuse, is relieved rather than Suggestive. Dietetic and drug tests. Macroscopic and chemical. Pain.

* A case showing a gradual transformation from the symptoms and findings of ulcer to those of terminal pyloric carcinoma has recently been under the author's observation. The distinctions while in general correct are by no means invariable (see gastric ulcer for further discussion).

†After a *test breakfast* the free acid which is the determining factor will exceed 50, the total acidity 70. The stomach is frequently entirely empty one hour after the meal because of coincident increased motor activity.

increased by taking nitrogenous food and the condition itself in most cases readily corrected by attention to diet and proper rest and recreation. Anæmia and other evidences of marked malnutrition are frequently absent but may result from limitation of ingesta. The appetite is frequently unimpaired, more often variable.

Anæmia.

Appetite.

CHRONIC HYPERSECRETION.—This includes two chief forms of hypersecretion usually marked by decided hyperacidity. In **Teichmann's disease** the increase of stomach contents in the fasting stomach is continuous. Though closely following the etiology of simple hyperacidity it is often associated with ulcer and *may* occur in pyloric stenosis, simple dilatation or chronic gastritis with impaired motility. It is a mere syndrome.

Continuous.

A mere syndrome.

Rossbach's disease, (nervous gastroxynsis) also frequently associated with gastric ulcer, may be merely a chronic form of what was primarily simple hypersecretion. The symptoms are those of the ordinary "bilious attack" with nausea, bilious vomiting, epigastric distress and *diffuse* pain and tenderness. It is recurrent, lasting from one to several days at a time and hardly deserves a place amongst actual diseases. Between the simple hyperchlorhydria and chronic hypersecretion stands the so-called "*alimentary hypersecretion*" which occurs only at the normal digestion periods, the contents showing often an excess of 50–200 c.c. over the amount of ingesta.

Recurrent.

Simulate surgical conditions.

Alimentary.

Comment.—Admitting that for the present the foregoing classification of hyperchlorhydria and hypersecretion must be preserved, the utmost caution is necessary in accepting it in the individual case, because of the frequency of unrecognized gastric ulcer and the tenuousness of the differential lines drawn. It is true however that in ulcer the pain and tenderness are more distinctly localized, *yet often both these symptoms and hemorrhage are entirely absent*, the ulcer going quietly on to spontaneous recovery or perforation. The crises of locomotor ataxia moreover may be associated with either hyper or hypochlorhydria.

Caution.

(3) **HYPOCHLORHYDRIA** (0.1—). Low HCl values point to subacute or chronic gastritis *but are frequently met with in early carcinoma*, certain late forms of *gastric ulcer, duodenal ulcer, dilatation* with or without stenosis, the early stage of *achylia gastrica* and also in *chronic disease of the gall-bladder, pancreas* and *vermiform appendix*. Its value as a positive symptom is therefore slight.

Indeterminate.

(4) **ANACHLORHYDRIA.**—As a symptom this is of positive and decided value, though met with in certain cases of advanced chronic

gastritis as well as nearly all gastric carcinomas, pernicious anæmia, and, in achylia gastrica and true atrophic gastritis, in which there is also an entire absence of the normal ferments. Unfortunately, according to the author's experience, many cases of dyspepsia apparently purely nervous, and especially those accompanied by marked malnutrition due to the common cause (viz.:—chronic starvation induced by foolishly restricted and monotonous diet), show this condition, the acid reappearing after a short period of treatment. The sign is therefore chiefly valuable in connection with the associated symptoms (see acute and chronic gastritis, cancer of the stomach and achylia gastrica).

Heterochylia.—This is merely a convenient term covering the cases of decided *intermittent variations* in secretion. It may occur in health or in any form of gastric disturbance, functional or organic.

GASTRIC NEUROSES AND CERTAIN PROMINENT GASTRO-INTESTINAL SYMPTOMS.—Aside from the hypersecretions previously discussed we may encounter *neuroses relating to pain, tenderness, peristaltic action, eructations, vomiting, motor spasm or relaxation, appetite and various paræsthesias.*

Appetite.—*Anorexia:*—Loss of appetite though related to many conditions may be described under this head. It is present in all fevers, in many chronic exhausting diseases and under the stress of violent emotion (grief, worry, anxiety), may be purely hysterical or neurasthenic and may take the form of mere loss of the hunger sense, actual repulsion, a sense of repletion, actual perversion, or, nausea upon taking a slight amount of food. Chlorosis, chronic alcoholism, hysteria, nervous and emotional overstrain, influenza and septic processes including advanced tuberculosis are some of the best known examples, but again the author would emphasize the frequency of chronic anorexia as the result of dietetic fads and fancies and narrowed monotonous dietaries.* Fool diets.

Bulimia.—An insatiable and inordinate craving for food is one of the marked symptoms in certain cases of true diabetes and occurs normally in convalescence from fevers such as typhoid and the exanthemata. It is met with as a *nervous or hysterical* manifestation as well as in cerebral disease, epilepsy, exophthalmic goitre, etc. In the hysteric and neurasthenic cases it may be associated with an absence of the sense of satiety (achoria) or there may be a craving for unusual, seemingly

* Again and again the most obstinate cases of dyspepsia with complete anorexia and excessive emaciation are promptly relieved under the rest cure and the gradual introduction of a varied and generous dietary.

or actually injurious, or possibly, disgusting articles, as is observed in hysteria, pregnancy, chlorosis and to a less degree in childhood, where it no doubt oftentimes represents in normal children a genuine structural need in so far as it relates to special types of ordinary food, the craving for sweets and fruits or the preference shown for an exclusive starchy or proteid diet being too often combated by parents.

Children's likings.

NAUSEA AND VOMITING.—May be either *central* or *peripheral* in origin, the centre being closely related to the respiratory centre in the medulla. Motor impulses to the diaphragm follow the phrenic nerve; to the abdominal muscles, the intercostals; while its sensory message follows the vagus. In gastric disease the act is distinctly reflex, in toxæmic conditions chiefly central. Purely psychic influences are capable of producing it as is seen in hysterical vomiting and that associated with profound emotion or mental shock. Its relation to special sensation is shown by the response to foul odors, disgusting tastes and shocking spectacles.

Nervous mechanism.

Toxæmic Vomiting.—The toxines may be bacterial as in the exanthemata, pneumonias, malaria, or severe infections in general, or non-bacterial, as in uræmia, diabetes, Addison's disease, profound anæmia or certain autointoxications. Furthermore one must consider the action of such drugs as apomorphia, ether and chloroform and the effect of deleterious gases and the overuse of alcohol.

Reflex and Central Vomiting.—As a precursor or accompaniment of apoplexy so also as a result of brain tumor, meningitis, thrombosis and embolism, vomiting may be a marked and somewhat suggestive feature. In anæmia both autointoxication and cerebral disturbance must be considered. It occurs in shock, collapse and occasionally in severe concealed hemorrhage, Ménière's disease, migraine and in the muscular insufficiencies and refractive errors seen by the oculist. Vomiting attending disturbances of equilibrium as typified by sea sickness is well known and in diseases of the abdominal organs the stomach is of course the most frequent cause of reflex vomiting whether there be actual inflammation, irritation, flatulence or mere disturbances of secretion. Under similar conditions it may attend ailments of related or neighboring organs such as the large and small intestine, the appendix, the rectum, liver, gall-bladder, pancreas, kidney and peritoneum. As regards the respiratory tract vomiting is readily induced by direct titillation of, or foul secretion adherent to, the naso-pharyngeal mucous membrane and may be associated with paroxysmal cough as in pertussis or pressure from mediastinal tumors. Circulatory causes aside from

Long list of causes.

anæmia and cerebral congestion or hemorrhage may be due to direct pressure, as in pericardial effusion, or a secondary congestion of the abdominal viscera in valvular or myocardial disease. Aside from the kidney the urinary tract is an infrequent source of vomiting but the genital tract furnishes many examples as in the vomiting of pregnancy, uterine displacement and ovarian or testicular disease.

Points to be Especially Noted.—These are (a) *frequency*, (b) *the time of day or night*, (c) *pain* and its persistence or intractability, (d) *associated nausea*, (e) *its relation to meals both as to the character of the food and the time elapsing after ingestion*, (f) *the character of the vomitus*, viz.:—amount, color, taste, the presence of remnants of remote meals, and blood bright red, dark or of the "coffee grounds" variety.

The Vomitus.—The general appearance of vomited material is too well known to need description and it is the significant variations that demand our attention. The amount, scant or excessive, suggests hyperacidity and excessive motility on the one hand, hypersecretion, dilatation or atony upon the other. *Vomitus from a fasting stomach if clear and of a sharply acid taste and odor suggests hypersecretion, if the mucous content be large and the tongue coated gastric catarrh is suggested.* Distinctly *bilious vomiting* is a common indication of acute indigestion but if persistent may also point to pyloric incompetence or duodenal stenosis, simple, or associated with ulcerative or malignant infiltration, to peritonitis, or, to intestinal obstruction. In the two latter conditions the vomitus soon presents an unmistakable fecal odor. *Mucus* may be intimately mixed with food residue and vomited at the outset as in gastric catarrh; is flocculent in hyperacidity and tenacious and stringy in anacidity. In hyperacidity it is soon dissolved and so usually points to sub or anacidity if present at the height of digestion. *Pus* may be visible in rare instances of gastric abscess or through fistulous communication with abscesses of other organs. In *cases of poisoning* the characteristic odor of the toxic substance may be evident and in cases of *stasis and marked fermentation*, butyric, acetic or valerianic acids may be detected. *In advanced ulcerative carcinoma* or in the presence of foul purulent material from whatever source the odor may be peculiarly putrefactive and in uræmia ammoniacal. Various parasites may be evident to the naked eye, such as round worms, thread worms and segments of tape worm. *The color* is, aside from food admixture and discoloration, yellow in anacidity, green from bile contamination or the development of certain yeasts, but is ordinarily dirty gray or greenish brown and, rarely, rose

Amount.

Bile.

Parasites.

Peculiar tints.

color from bile decomposition with high acidity or in the crisis of tabes dorsalis (Lorenz) with hypersecretion. *Bile* may be present in any vomitus if the efforts are prolonged. Occurring at the outset or in repeated slight attacks it strongly suggests pyloric or duodenal infiltration, in the latter case *pancreatic secretion may be demonstrated by the digestive activity of the vomitus in alkaline media.*

"Coffee grounds." Clots.

Hæmatemesis.—If the blood be recently effused it is bright red and fluid, if partially digested it resembles coffee grounds, if in an intermediate stage and in sufficient quantity it may be clotted.

How simulated.

The appearance of blood is simulated by jellies and jams, cocoa, coffee, beef juice, such drugs as iron and bismuth, the dark red wines, grape juice and fruits such as currants, cranberries and cherries.

Amount.

The amount of blood may be large or small and the small amounts may be derived from any source, indeed a mere streaking of mucus may accompany excessive and persistent vomiting and indicate no serious lesion. On the other hand, hemorrhages of all degrees occur in ulcer and carcinoma, splenomegaly, certain cachetic conditions of the hemorrhagic type such as advanced leukæmia, hæmophilia, scurvy and purpura, virulent infections chiefly associated with tropical fevers, such as yellow fever and pernicious malaria and, the action of irritant and corrosive poisons. Blows and wounds, the varices of portal obstruction, chronic heart disease and vicarious menstruation must be considered.

Sources of Error.—In nose bleed, hæmoptysis, ruptured tonsillar abscess and similar conditions the blood may be swallowed and its source may be recognized only through the presence of the causative factors named.

Hæmoptysis.

In hæmoptysis, the blood is bright red, frothy and of alkaline reaction; there are usually physical signs, cough and often distinctly localizing subjective sensations.*

Stools or stomach contents.

Chemical and Microscopic Tests for Blood.—The following test will apply equally to blood in the stools and in the stomach contents and it must be remembered that an examination of the stools is of the utmost importance in all cases of supposed carcinoma or gastric ulcer, vomiting being frequently absent and slight hemorrhages unrecognized.

Weber's Test demands that a portion of the suspected material placed in the test-tube be treated with several drops of strong acetic acid and shaken up thoroughly with one-third its volume of ether, the supernatant ether is pipetted off and if necessary cleared by adding

* Though in hæmoptysis the blood is raised by coughing, vomiting is a frequent accompaniment and may predominate, the cough being slight. Hence too much stress should not be laid upon this as a differential factor.

a few drops of alcohol and the solution should then show an absorption band* of acid hematin by the direct vision spectroscope. *Guaiacum Test.*—To the ethereal extract may be added 10 drops of freshly prepared tincture of guaiac and double the amount of old turpentine, blood being indicated by the bluish or purple color, which can be extracted and better defined if shaken up with chloroform. The alcoholic extract of Barbadoe's aloin may represent the tincture of guaiac in this test and is less liable to disturbance from free HCl. Best method.

Teichmann's Test requires the careful evaporation of the suspected material on the slide after which a crystal of sodium iodide or sodium chloride is added, followed by a drop of glacial acetic acid. Too rapid evaporation is prevented by the use of a cover-glass and it is then gently heated to the exact boiling point, fresh acid being added as evaporation goes on. When a brown color appears, indicating the formation of hemin, complete evaporation is allowed and a drop of water run under the cover-slip. The microscope will then show the characteristic black or brown rhomboidal crystals of hemin. Iron causes no disturbance of the *chemical tests* for blood. Valuable test.

Microscopic Findings.—The food detritus depends upon the character of the meal previously taken and the time elapsing since its ingestion, but, in general, consists of muscle fibres and starch granules more or less changed by cooking and digestion, the former being recognized by their markings, the latter by their peculiar form and structure or their blue reaction with Lugol's solution. Skins of fruit, seeds of berries or even more digestible substances remaining from remote meals point to impaired motility or actual stenosis, saliva is indicated by pavement epithelium and the so-called salivary corpuscles and the various vegetable cells are usually so peculiar and bizarre as to at once suggest their nature. Fat may appear in the form of crystals or of the highly refractile droplets soluble in ether, staining orange red with Sudan III and black with osmic acid (1%). The presence of a few red blood corpuscles after vomiting or the passage of a stomach tube is not necessarily pathologic. Mucus may be present in all degrees according to the nature of the lesion but is rarely absent entirely even if HCl be present and may take the form of snail like masses. Such mucus is likely to be from the buccal cavity or esophagus.

Starch and muscle fibres.

Indigestible residue.

Saliva.

Fat.

Microscopic blood.

Mucus.

*The absorption spectrum consists of one strongly defined band in the red and three less defined or often entirely absent. (One in the yellow, one between the yellow and the green and another between the green and the blue.)

Boas-Oppler vs. Leptothrix.

Vegetable Parasites.—The Boas-Oppler bacillus, the sarcina ventriculi, the leptothrix buccalis and the tubercle bacillus are the most important. In carcinoma the Boas-Oppler bacillus is present in the form of large long rods frequently arranged in chains and staining brown with Gram's solution, whereas the leptothrix which resembles it stains blue. It not only indicates large amounts of lactic acid but *is present in 80% or more of the cases of cancer of the stomach and nearly always absent in simple dilatation or benign stenosis.* The sarcinæ on the contrary are present in large numbers in these benign cases though rarely in acute or chronic gastritis, simple atony, ulcer and cancer of the stomach.* Both the large and small variety appear usually as if arranged in bales. Yeast fungi are present in large numbers in marked degrees of atony or ectasia and, in high acidity, unchanged starch granules will be plentiful, and in subacidity the muscle fibres will be correspondingly numerous in their definite and unchanged form. The Boas-Oppler bacilli usually abound in the larger clots sometimes met with in cancer.

Yeasts.

Cancer Cells.—Rarely one finds detached particles of new growths but more often such fragments are absent or unrecognizable.

"Heart Burn," ("Pyrosis" or "Water Brash") covers a regurgitation of stomach contents common in many forms of gastric disturbance, and *merycismus* (rumination), usually observed in neurasthenia, hysteria, idiocy or epilepsy, covers *voluntary* regurgitation and remastication of the food.

Peristaltic Unrest (supermotility, hyperkinesis).—This troublesome excessive motility, is accompanied by noisy gurgling and rapid emptying of the stomach, but may occur in the empty organ. It is a common transient phenomenon but may be persistent in hysteria and neurasthenia.

Eructation (pneumatosis, belching, flatulence).—This common phenomenon needs no explanation. Ordinarily negligible, it is a persistent, troublesome and characteristic symptom in certain nervous dyspepsias associated with marked neurasthenia or hysteria. The term "*pneumatosis*" is properly applied to the condition of extreme distension of the stomach by air swallowed by certain dyspeptics or by malingerers.

Often misleading.

Gastric Spasm.—Spasm may occur either at the cardia or pylorus and both may be purely nervous phenomena, the former almost invariably so, the latter being most frequently associated with hyper-

* They are reported by Heichelheim as present in small numbers even in the carcinomatous stomachs *showing high acid values.*

acidity; in either there may be a subjective localized sensation of obstruction, both may be painful and the latter may produce visible or even reversed peristalsis and, if long continued, lead to actual dilatation. If the two conditions are simultaneous (invariably neurotic) the marked overdistension may be evident.

Gastric Hyperæsthesia.—Marked gastric intolerance with a sense of weight, fulness or burning and without an organic basis may be encountered in hysteria, neurasthenia, exhausting and debilitating disease, mental and physical overstrain, profound anæmia, shock, and forced or voluntary fasting for long periods. It may be associated with marked diffuse tenderness corresponding to the stomach outline and is probably the chief cause of symptoms in hyperchlorhydria.

Gastralgia has been previously referred to as a misused term representing in most instances appendicitis, gall stones, gastric ulcer or cancer, the gastric crises of locomotor ataxia, splanchnic arterio-sclerosis or angina pectoris. A true neuralgia of the stomach is exceedingly rare. A rare condition.

NERVOUS DYSPEPSIA (combined gastric neuroses).—These terms, of which the second is the more descriptive, cover certain forms of gastric disturbance *prolific in subjective, barren in objective symptoms* and commonly associated with nervous overstrain, shock, grief, worry, frank neurasthenia or hysteria, or, due to reflex causes such as chronic appendicitis, pelvic disease, and movable kidney. *It is essentially a mere syndrome.* The tongue is usually clean or lightly furred and may show some swelling and lateral indentation. When the stomach is empty the patient may complain of faintness and a "gone" sensation in the epigastrium, headache, or a sense of cerebral pressure or dulness, marked subjective weakness and palpitation after taking food. There is troublesome distension, eructation and oftentimes peristaltic unrest associated with pyrosis and irritating borborygmi. Dizziness, epigastric pulsation and a globus hystericus may be encountered. The gastric contents are usually normal but may show hyper-, hypo- or a temporary anacidity. Heterochylia is common, and malnutrition may or may not be marked, Persistence of symptoms is due to an unduly restricted or monotonous diet, lack of rest (mental and physical) proper recreation and diversion and a pernicious and persistent form of introspection. *Many of these cases require absolute rest, isolation, a gradually increased and ultimately generous diet, absolute control by a specially trained nurse, the oversight of a competent physician and a modicum of common sense and will power as a leaven.*

A neurosis.

Tongue.

Suggestive symptoms.

"Globus hystericus."

Heterocylia.

Bad diet.

Introspection.

ANOREXIA NERVOSA.—This curious condition characterized by absolute loss of appetite and frequently by abhorrence and rejection of all foods, may follow prolonged fasting (starvation "cures"), shock, grief and the like, or, *as is usually the case, be associated with hysteria or profound neurasthenia.* In hysterical cases especially, the patient may be profoundly anæmic, will *promptly* vomit all food administered, complain of vague and often shifting abdominal pain and tenderness, and frequently pretend to vomit blood, or, if posted, imitate the painful crises of ulcer. Emaciation may be extreme and a fatal issue is possible if proper treatment is not available. Absolute rest, isolation, and firm control by a competent nurse and physician will usually clear up the diagnosis and cure the patient. One of the most characteristic features is the *almost instant vomiting of the food* though usually water will be retained and frequently the act is one of mere regurgitation.

May be fatal.

ACHYLIA GASTRICA SIMPLEX (apepsia).—This interesting ailment or syndrome is of unknown causation and affects adults almost or quite exclusively. *So long as intestinal digestion remains unaffected few or no symptoms* are produced yet an examination of the stomach reveals complete loss of HCl and digestive ferments and a diminished secretion of fluid. There is no pain, the fasting stomach is empty and one hour after a test breakfast the residual content is small but wholly unchanged and lacking mucus or any sign of fermentation. Microscopically one finds unchanged proteid and advanced starch digestion (ptyalin in the absence of HCl. Fehling's test will be positive, the iodine test for unchanged starches negative. Motility is increased as might be expected and the intestinal juices complete the process, In advanced cases the intestine may become the seat of irritation and catarrhal inflammation and the stools show imperfect digestion of the proteids. In such cases indicanuria is marked. In genuine simple achylia gastrica the mucous membrane is not atrophied but is readily injured and abraded.

Remarkable findings.

No gastric digestion.

Increased motility.

GASTRIC ATROPHY (achylia gastrica with atrophy).—The first term is preferable and should be limited to those cases of anachlorhydria and apepsia in which atrophy is secondary to no other ailment, such as carcinoma *local or remote*, and especially fits cases presenting the blood picture of pernicious anæmia, though this symptom may in certain cases be lacking.

Diagnosis is assisted by the following factors:—dyspeptic symptoms and even *pain* may be marked, indeed anorexia or aversion to meats may occur exactly as in carcinoma, but, unlike the latter, the fermen-

Misleading signs.

tation acids are absent and there are no evidences of stenosis or dilatation. Intestinal symptoms may supervene and the usual anæmia may assume any grade. The picture then is usually one suggesting either carcinoma or pernicious anæmia but lacking the lactic acid and Boas-Oppler bacilli and the evidences of stasis and dilatation so common in carcinoma. *High grade anæmia superadds its own symptoms.*

Anæmia perhaps extreme.

Symptomatic Hypochylia and Achylia.—To the foregoing must be added the purely secondary diminution or complete absence of HCl and the ferments. It may play a part in the cachexia of terminal nephritis, diabetes and tuberculosis, is not infrequent in severe chronic catarrhal gastritis with atrophy or the anachlorhydric form of gastric ulcer and is present in 80% of the carcinomata, *local or remote.*

A remarkable fact.

Summary.—Achylia gastrica may be (a) *symptomatic or secondary*, (b) *associated with primary gastric atrophy or* (c) *occur as a definite syndrome without marked* subjective disturbance being *almost symptomless save for the gastric findings unless intestinal irritation supervenes.*

Three types.

Diagnosis.—No diagnosis should be made of either the atrophic or simple form until all possible primary sources have been excluded. The atrophic form is suggested by symptoms of carcinoma, erosion, or profound anæmia, but lacking certain of their gastric findings; simple achylia by the astonishing lack of severe symptoms with an entire absence of proteolytic agents.

Seek a primary cause.

DISORDERED MOTILITY.—*Hypermotility* has already been touched upon and is marked in hyperacidity and present in achylia, some cases of diabetes mellitus, tape worm and the ingestion of certain drugs such as pilocarpin and strychnine.

Motor insufficiency may be due to actual atony and the term is less properly applied to the stasis produced mechanically by pyloric stenosis as in these cases there may be actual hypertrophy of the muscular layers and, for a time at least, partial compensation. Dilatation results from the stasis of either myasthenia or stenosis.

Atony vs. Stasis.

GASTRIC ATONY.—An attempt to sharply classify the various conditions associated with muscular insufficiency or actual dilatation of the stomach leads to much confusion and *it is better to consider the stomach and its musculature exactly as one would the heart. Either organ may be abnormally large yet perfectly competent.* In the case of the stomach this interesting but symptomless condition is known as *megalogastria. Weak musculature resulting in lessened motor activity and relaxation of the muscular walls constitutes true atony of the stomach and as in the case of a chronically weak heart leads to lessened*

Megalogastria.

effective functional activity. Finally, if an actual obstruction exists at an exit, the stomach and the heart act alike in that they try to overcome, by increased action and hypertrophy of the muscle, the mechanical obstacle or, if this is not possible, to undergo passive dilatation with consequent stasis. The parallel is quite exact and helps to clear up this subject which is involved in a maze of contradictory opinions even at the present time. Disregarding megalogastria we have first to deal with gastric atony or myasthenia and its inevitable accompaniment, decreased motility from "muscular insufficiency." This may exist without marked or permanent dilatation in any degree, but *persistence* of muscle weakness *combined with prolonged retention of the stomach contents of itself tends to produce a secondary dilatation* (ectasia) varying greatly though less commonly reaching an extreme form than is the case in actual mechanical obstruction. Hence we find that in *atony* considerable degrees of dilatation are possible without the extreme manifestations of gastric stasis and fermentation encountered in pyloric stenosis. *In this last condition* as in stenosis of the aortic orifice of the heart unless myasthenia or atony pre-exists there is primarily an hypertrophy of the muscular coats which may for a long time prevent extreme dilatation or marked stasis. Ultimately, however, either because the stenosis becomes more extreme or the muscular weakness predominates over hypertrophy, marked dilatation occurs with an increased degree of myasthenia and resulting stasis.

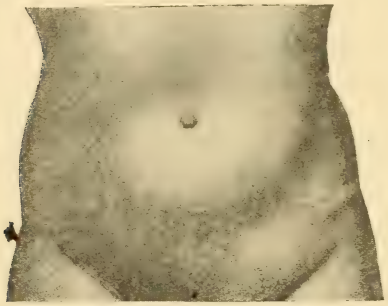


Fig. 116.—Dilated stomach. Combined displacement and dilation of lesser degree. (After Riegel.)

Ectasia.

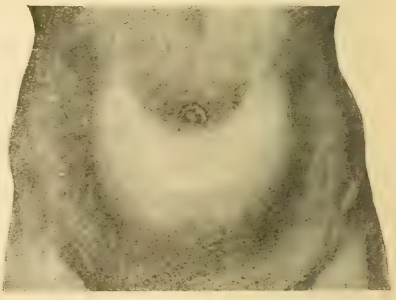


Fig. 117.—Dilated and displaced stomach (crescentic form). Outline—personal observation. (Figure after Riegel).

Stasis.

Gastroptosis.—Any case of dilatation if marked *may* be associated

with varying degrees of displacement (ectasia with gastroptosis) but marked ptosis may exist without dilatation (gastroptosis proper). Having the preceding factors in mind, one may proceed to discuss the conditions under three chief heads.

Ectasia.

(1) **Atony** (myasthenia, "relative", "motor" or facultative insufficiency).—This covers the *non-obstructive* form and may be primary, secondary, acute or chronic and associated with merely temporary dilatation or abnormal distensibility, or, true ectasia. *In rare instances acute, oftentimes fatal dilatation occurs*, a condition of special importance in connection with heart disease, arterio-sclerosis, the acute infections and major operations, particularly of the abdominal type. **Chronic dilatation** of an extreme form may be seen in the employes of breweries or others who are gross eaters and heavy beer drinkers. No other disease is richer in *etiologic factors*, amongst which are heredity (it may be congenital), nervous shock, grief, worry, mental or physical overstrain, exhausting diseases, overeating and drinking, epilepsy, migraine, brain injury, abdominal traumatism, overuse of tobacco and alcohol, and finally, nervous dyspepsia, hepatic cirrhosis, pancreatitis, cholelithiasis, chronic constipation, chronic appendicitis and certain acute infections (typhoid, diphtheria, scarlatina, etc.).

Chronic or acute.

Etiology varied.

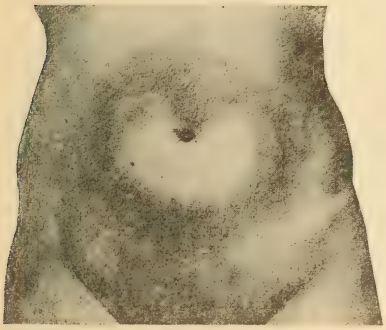


Fig. 118.—Simple Gastroptosis. Personal observation. (Figure after Riegel.)

The Symptoms.—*Subjectively*, the symptoms *are those described under nervous dyspepsia save that in many cases of atony the patient is symptom free when the stomach is empty*. The slightest quantities of food give the sensation of *repletion* and *distension* and there is *obstinate* constipation. *Objectively*, one notes *abdominal distension* and oftentimes visible gastric enlargement with or without gastroptosis yet *the milder cases may show merely excessive distensibility, as revealed by inflation, or the low level of dulness resulting from the ingestion of a pint of water*. The gastric outline is usually markedly extended to the right as well as downward and contrary to the findings in stenotic cases, *visible* or *palpable peristalsis is lacking or slight*. *Succussion, both superficial*

Inefficient musculature.

Distensibility or ectasia.

Salol test.

and deep, is apparent even 6 or 7 hours after a meal and is abnormally low in position. *Salol excretion* (see page 241) is delayed and *residual food* will be found 7 hours or more after the Riegel dinner, though usually absent if the meal *has been retained over night.* If the case be watched from day to day marked variations in the size of the stomach may be noted, and, finally, *the stomach contents may show a relatively slight degree of stasis as indicated by fermentation*, though the stomach findings vary greatly and are not in themselves distinctive.

Ultimate slight stasis.

Acute Atony.—Terrifying epigastric oppression, violent dry retching, or, more rarely, slightly productive vomiting, actual pain, and belching followed by persistent gaseous distension, cold extremities and a weak and rapid pulse, mark an ailment less often recognized than it should be as a cause of severe illness and relatively sudden death.

A cause of sudden death.

Fig. 119.—Dilated stomach, combined displacement and dilation. Slightly modified to conform to personal observations. (After Riegel.)

Post-stenotic Motor Insufficiency.—Here actual obstruction *not mere loss* of muscle tone is the chief factor, indeed, actual muscular hypertrophy may be present in a dilated stomach as in the case of the heart with valvular incompetence.

Obstructive in type.

Etiology.—Whatever causes produce obstruction of the pylorus or duodenum may cause gastric dilatation and insufficiency. It may follow operations upon the stomach, duodenum or gall-bladder, pyloric tumors or hypertrophy, congenital or acquired,* pyloric spasm (see p. 253) and obstructive scars or deformities. Even tight corsets or plaster jackets may produce chronic obstruction and dilatation.

Various causes.

Symptoms.—*The symptoms of simple nervous dyspepsia or myasthenia predominate but pain may be present* and be severe, paroxysmal and *attended by violent visible peristalsis or palpable contractions*, or, on the other hand, it may be absent or replaced by or associated with

* Congenital stenosis is far commoner than has been supposed. Three cases (Drs. J. U. Goodrich, P. A. Hoff and W. Ramsey) having been brought to the author's attention within 90 days; all confirmed by autopsy.

extreme epigastric oppression or distension with or without tenderness.

Objective symptoms are readily elicited. *The stomach is often visibly distended or if inflated appears in characteristic but enlarged outline with or without displacement* (gastroptosis) and is readily palpated. Increased resistance may be noted as well as rhythmically intermittent rigidity. *Peristalsis* may or may not be visible but is most marked during painful crises, if such are present, or shortly after a meal. As in the heart, long persistence of stenosis may cause loss of muscle tonus and diminution of palpable contractions or visible peristalsis. The degree of dilatation may be determined by inflation and auscultatory percussion, palpating the stomach tube *in situ*, fluoroscopy, transillumination, the gyromele, etc. (see p. 235). *Changes of note corresponding to changes of posture* may be evident as in pulmonary cavities if fluid be present. *Succussion is of course constant* in marked ectasia. *Residual contents* in marked quantity, varying with the degree of stenosis and ectasia, *are found in the fasting stomach* even though the Reigel dinner be taken the preceding evening after lavage, and the contents are foul smelling (garlic or cabbage or hydrogen sulphide) and if placed in a fermentation tube with glucose (Einhorn's tube or an inverted test-tube) will within 24 or 48 hours show at least $\frac{1}{20}$ volume of displacement by gas and the development of yeast plants. Indeed fission fungi will abound if fermentation be present, and indicate persistent stasis.

Usually frank.

Muscle tonus.

Stasis signs.

Pain and Vomiting.—Pain is variable, often absent save in slight degree, or is substituted by extreme distension and oppression. *The vomitus is* as above described, *frothy, of excessive quantity* and *often contains remnants* of *several previous meals*, especially seeds and the like. It frequently separates into three layers on standing and is bitter and foul to the taste. *The total acidity is increased by fermentation acids and the microscope shows in benign cases the yeasts and sarcinæ; in cancer, the Boas-Oppler bacilli.* The fatty acids may be present by chemical and microscopic tests, the urine is scant, concentrated if vomiting be profuse, and if gastric acidity be high is often alkaline in reaction. *The persistent presence of bile in quantity invariably points to duodenal stenosis.*

Distension.

Vomitus.

Acidity.

Urine.

Complications.—Acute autointoxication occasionally occurs and even coma may result. Extreme distension may not only produce dyspepsia, bradycardia or palpitation but may seriously endanger a weak heart. Gastro-succorrhœa is common and, finally, that rare and

Autointoxication.

interesting condition known as *gastric tetany* may supervene, during the passage of a stomach tube, vomiting, or even percussion.

Distinctive signs of stasis.

Differentiation.—*The clinical distinction between these various forms rests primarily upon the evidence of delayed emptying of the stomach and the presence and degree of stasis as indicated by persistent retention of food residue and fermentative changes.* Furthermore in actual mechanical obstruction *recurrent and copious vomiting*, showing persistent and often ancient food residue, *marked visible or palpable peristalsis* and usually the *presence of a pyloric tumor* assist the diagnosis. In *stenotic* insufficiency, moreover, we are ordinarily dealing with carcinoma, more rarely with pyloric ulcer or its scars, and *the gastric contents will vary with the causative factors.*

Benign vs. Malignant.

As between the benign and malignant cases great difficulties may arise in diagnosis but in general it may be said that, while marked stasis characterizes both, the presence of large numbers of the Boas-Oppler bacilli, the cachexia, profound anæmia and the entire absence of hydrochloric acid, free and combined, so nearly constant in cancer, are absent or present in less degree in benign stenosis, in which moreover, the sarcinæ are present in large numbers.

Gastroptosis.—The general topic of enteroptosis (p. 287) covers this condition of which it is a part. Gastroptosis may exist with or without dilatation, and may be symptomless or assume the character of neurasthenia and nervous dyspepsia, or, motor insufficiency, though the *latter is rare save in the severer forms.* Amongst the commoner symptoms are:—subjective distension, troublesome borborygmi especially if tight clothing or bands are worn, tenderness over the solar plexus, subjective aortic pulsation and, in the presence of movable kidney, a dragging sensation or dull pain referred to the region of the sacro-iliac joint or lower lumbar region. Succussion is often present and low in site. The displacement is detected by the low position of the lesser curvature readily determined by inflation, as is any coincident dilatation.

HOUR-GLASS CONTRACTION (*bilocular stomach*).—This rare deformity if not congenital, results usually from the healing of a large annular ulcer, more rarely from adhesions or scirrhus. The stomach is divided into two lateral segments and the communicating opening may be relatively large or very small. The symptoms vary with the cause and may closely simulate pyloric stenosis if the opening be small.

Usually secondary.

Diagnosis.—Inflation alone will rarely make the diagnosis. The following signs are valuable:—(1). The return of only the major por-

tion of a litre of water introduced by the tube. (2). In distension by air or liquid, if the opening is narrow (a) the bulging and dull percussion note may at first be only in the cardial pouch and *later* be apparent in the pyloric portion. (b). The water or gas may be heard or felt to pass through the narrow orifice especially if pressure be applied. (c). Transillumination or fluoroscopy (bismuth and porridge method) will show only the cardial pouch. (d). If all recoverable water be withdrawn succussion will be evident only over the *pyloric loculus*. Signs distinctive.

ACUTE GASTRITIS (gastritis glandularis acuta, acute gastric catarrh, phlegmonous gastritis).—**Definition.**—An acute (simple, toxic, or phlegmonous) inflammation of the gastric mucosa.

Etiology.—Indiscretion in diet, nervous shock, toxic irritants such as ptomaines or arsenic, alcohol, mercuric chloride and potassium cyanide, or, if phlegmonous, pyæmia, septicæmia, the exanthemata, and rarely direct infection. It is most frequent at the extremes of life and in those debilitated and dyspeptic. Excessively hot or cold drinks, overeating and *imperfect mastication* are important factors.

Morbid Anatomy.—The lesions vary from a simple catarrhal inflammation to necrosis and single or multiple abscesses.

Symptoms.—Mere subjective weight or fullness, anorexia, and, a coated tongue with or without nausea, slight pain and general dyspeptic manifestations may be the only symptoms. In the severer forms one finds headache, epigastric pain, nausea, vomiting and constipation or diarrhœa, complete anorexia, excessive thirst and a burning sensation. In the toxic forms, prostration is extreme, the pulse feeble, the surface cold, the case tending to coma and death. Such cases may occur in myasthenia and stenotic ectasia. Severity varies.

Stomach Contents.—Mucus is present in the vomitus, associated with blood and shreds of mucous membrane in the toxic forms and perhaps pus in the phlegmonous forms. Free HCl is usually absent.

Prognosis.—Recovery is prompt save in exceptionally severe toxic or phlegmonous cases.

CHRONIC GASTRIC CATARRH (chronic gastritis).—A chronic catarrhal inflammation of the gastric mucosa. **Etiology.**—The disease may follow an acute gastritis, but is more frequently associated with a monotonous or improper dietary, chronic diseases of the heart, liver, kidneys and lungs, gout, diabetes and the anæmias, excessive smoking and drinking, the use of highly spiced dishes, rapid and excessive eating and imperfect mastication. It follows certain infectious fevers as typhoid, cholera, yellow fever and the exanthemata, and, lastly, ulcer. Usually secondary. Primary lesions may dominate.

cancer or dilatation of the stomach itself. It follows that certain complicating cases are hardly distinguishable as such.

Common signs.

Symptoms.—Amongst the commoner are:—flatulence, belching, morning-malaise, nausea and perhaps vomiting, pyrosis, foul taste in the mouth, vertigo, palpitation, dyspnœa, cough and a poor or variable appetite often associated with a sense of repletion after even a light meal. *Morning vomiting* occurs especially in the chronic gastritis associated with hepatic cirrhosis ("drunkards' catarrh"). Constipation in some cases alternating with diarrhœa is the rule, there are usually evidences of a poor circulation, the tongue is coated and marginally indented by the teeth, and, nervous irritability is common. *Marked loss of weight is seldom seen* save in distinctly secondary cases, where it is usually due to the primary cause.

Stomach Contents.—In advanced cases *free HCl is diminished or absent, total acidity below normal, total stomach content usually increased, mucus abundant* even in the empty stomach, and the *digestive ferments* diminished.

Limitations.

Differential Diagnosis.—All differential diagnosis in diseases of the stomach is subject to limitations and exceptions, it being often impossible to draw hard and fast lines, and chronic gastritis must like all others be encountered in its early and complicating forms, lacking in the one case its most characteristic symptoms and in the other blending with or overshadowed by the primary disease. *A chronic gastritis lacks the severe pain, extreme localized tenderness, hæmatemesis and high HCl values of ulcer* and the *marked emaciation, lactic acid, Boas-Oppler bacilli and tumor so generally present in cancer. Achylia gastrica lacks both HCl and ferments, and, in the neuroses, the neurasthenic or hysteric symptoms, the more trifling or unlike disturbances of gastric secretion and the absence of an excess of mucus usually differentiate it.*

Prognosis.—Varies according to the cause. Many cases may be entirely cured, others never.

GASTRIC ULCER.—Definition.—An ulcer of the gastric mucous membrane, usually single but often multiple and tending to perforation.

Etiology.—Aside from direct irritation or traumatism hyperacidity and antecedent anæmia appear to be the most important *primary* factors, but the true cause is not known.* *Sex.* About 60% of the

* Recent investigations indicate the existence in the normal stomach of protective anti-enzymes but under what conditions their activity fails is as yet unknown.

cases appear in females. *Age.* It occurs most commonly between the ages of 20–40, least often in children and elderly persons, and, there is probably no hereditary element. *Occupation.* Direct injury as from blows, tight lacing, pressure; the ingestion of corrosive drugs and such occupations as lead to anæmia, irregular or hasty eating and the use of improper food are important factors.

Young females chiefly.

Varieties.—(a). *Common form,* of pronounced and definite symptomatology denominated by Welch "gastralgic-dyspeptic." (b). *The latent forms. Single or multiple ulcers may exist for years and perforate or spontaneously heal without localized symptoms.* (Probably 50% of ulcers are unrecognized clinically.) (c). *The acute perforating form.* In this there is a rapid advance and perforation and death may occur with few or no *premonitory* local symptoms. (d). *The acute hemorrhagic form.* In this the symptoms are usually vague, of brief duration or may be entirely absent until profuse hemorrhage occurs.* Other types occasionally given are apparently unimportant.

Often overlooked.

Treacherous types.

Pathology and Morbid Anatomy.—The ulcers vary in size from mere erosion to necrotic areas measuring from 4 to 6 inches in diameter. They are ordinarily small, ½ to ¾ in. round, and clear cut with little or no induration of the base or the margins, though if chronic they may be irregular or sinuous with terraced sides and marked infiltration. They are ordinarily single, not infrequently there are from three to five, very rarely a score or more. *Location.* Ulcer commonly occurs on the posterior aspect of the pyloric end of the stomach near the lesser curvature, from 10–15% are found at the pylorus,† and these if chronic may lead to pyloric obstruction and gastric dilatation. They are rare at the cardia and greater curvature. Gastric ulcers show a marked tendency to recur, usually within an old scar of a healed ulcer, and in an old ulcer base true carcinoma may develop. *Healing.* They heal by granulation and in cases of extensive ulcer may produce deformity such as the hour-glass contraction but more commonly a pyloric stenosis. *Perforation.* Perforation may result in general peritonitis demanding surgical interference and tending to a fatal termination, or, adhesions may form limiting the spread of the infection and saving the life. Many such cases of perforation go unrecognized, the omentum, liver and gall-

Usually single.

Rare at cardia.

Recurrence.

Self limitation.

* In one such case coming under the author's observation a young girl who had not had even a symptom of dyspepsia was seized suddenly with so profuse a hemorrhage as to cause collapse and cover the floor of a small kitchen with a layer of bright arterial blood.

† Mayo's figures (75%) are of course those of a surgical clinic to which this type would naturally drift.

Misinterpretation.

bladder and the pancreas may be involved, and, such adhesions complicate a clinical picture and lead to mistaken diagnosis. The author has observed cases in which the gall-bladder was involved, the symptoms exactly simulating gall stones. The acute perforations are usually

Obscure abscesses.

direct and involve the general peritoneal cavity. Obscure abscesses may result in the chronic forms, subphrenic abscess and pyopneumothorax being sometimes observed and fistulous communication may involve tracts far distant from the original seat of trouble. The frequency

Fallacious figures.

of perforation as usually stated (13 to 18%) is certainly too high, being necessarily based upon cases yielding positive symptoms or coming to autopsy, and of course omitting the doubtful or larval cases which, no doubt, outnumber the others.

Diagnosis.—The diagnosis of gastric ulcer still largely depends upon the concurrence of the old tetrad of symptoms, *pain, localized tenderness, vomiting and hemorrhage*, yet in many instances we meet and recog-

Larval cases.

nize early, latent, or larval cases which lack these franker symptoms.* A large proportion demand the careful balancing and analysis of a large number of factors and *fatal perforation or hemorrhage may occur in cases which have previously lacked the symptoms necessary to even a tentative diagnosis.* Placing the determining factors somewhat in the order of their clinical importance we have (a). *Pain.* (b). *Localized tenderness.* (c). *Hemorrhage.* (d). *Vomiting.* (e). *High hydro-*

Important factors.

chloric acid values. (f). *General dyspeptic symptoms.* (g). *Anæmia.* (h). *Age.* (i). *Sex.* (j). *Occupation.*

(a). **Pain.** The characteristics of the pain of gastric ulcer are (1st). *Its intermittent and paroxysmal nature, and its relation to*

Characteristics.

the taking of food and to special forms of ingesta. (2nd). *Its time of occurrence.* Being dependent primarily and chiefly upon

Cause.

the increased secretion of hydrochloric acid incident to the digestive process, it is not as a rule immediate but usually becomes

Incidence.

severe and even cramp like in from half an hour to two hours

Duration.

after eating and *continues* until the stomach is emptied by vomiting, stomach washing, or by the natural mechanism. In some instances distress almost immediately following the ingestion of very hot, cold

Helpful sign.

or acid substances is significant. Only in exceptional cases, namely those of continuous hypersecretion do we find persistent pain of a duller

Persistent pain.

character between meals and especially at night. This fact suggests the importance of stretching and distension as well as peristalsis in the production of crises. The *location* of the pain necessarily varies

* The disease is often one of extremely slow and insidious development.

somewhat with the position of the ulcer and of the stomach itself, being as a rule lower in tightly laced women and in cases of ectasia than under ordinary conditions. As five-sixths of the stomach lies to the left of the linea-alba, and most ulcers are upon the lesser curvature, *one usually finds the pain at, or, more commonly, just to the left of the median line between the ensiform and the umbilicus, usually near the former.* The pain itself varies greatly in *character and degree. In its typical form it is a boring deep seated pain,* but is also described as *burning, stabbing, cutting or cramp-like*, quite frequently felt at the back, sometimes radiating upwards to the region of the shoulder blades. This typical pain is often replaced by that of a duller character and a suggestive feature when present is the relief accompanying changes in attitude both in the presence and absence of adhesions. Increase of pain in recumbency suggests but does not prove a lesion of the posterior wall, and a similar inference applies to the lateral or prone positions as regards a lesion of the pylorus or anterior wall. **Localized tenderness.** The importance of this sign lies in the fact that a distinctly circumscribed superficial or deep tenderness, *often extreme*, is usually present and corresponds closely to the area of maximum pain. It is usually epigastric but is a trifle *to the left* of the median line and *localized* as contrasted with the tenderness of gastric hyperæsthesia which is diffused over the whole outline of even the inflated stomach. About ⅓ of the cases show a sharply localized area of superficial or deep *dorsal tenderness*, often extreme, slightly to the left of the lower dorsal spines (3–4 cm.)*.

Important points.

Referred pain.

Posture.

Usually present.

Site.

Spinal areas.

Hemorrhage.—Hemorrhage which occurs in ⅓ to ½ of *recognized* ulcers is, if large, always associated with vomiting and is easily recognized as of gastric origin; but the smaller hemorrhages rapidly undergo such digestive changes as to closely simulate the small dark clots or coffee ground particles of carcinoma. Furthermore, severe or even fatal hæmatemesis may be due to gastric erosion or hepatic cirrhosis as well as ulcer. The stools as well as the stomach contents should be carefully investigated before declaring hemorrhage absent and concurrent symptoms be carefully weighed before a frank hemorrhage is ascribed to ulceration. *Enormous hemorrhage may be recovered from and but a small number die directly from it.* **Vomiting.** Though often coincident with a painful crisis vomiting with or without hemorrhage is far from constant and nausea is more often seen. If present, it is usually irregular and in any event relieves or modifies the pain.

Usually evident.

Disturbing factors.

Caution.

Inconstant.

* Rarely it is bilateral or on the right side alone. A second zone may be found occasionally at the level of the fourth and fifth dorsal spines.

Hyperacidity.—High hydrochloric acid values are *common* (30–45%), normal values probably *the rule*, subacidity the exception.* As hyperchlorhydria is a common condition without ulcer, so that the symptom while important is only *corroborative*, nevertheless *decided hypoacidity goes far to exclude ulcer, and lactic acid is almost invariably absent.* (Subacidity is usually associated with complicating catarrhal gastritis or ectasia.) The vomiting in hyperacidity is sharply acid to the patient's taste, and it must be remembered that the proper determination of high hydrochloric values should be based upon a test dinner rather than the meagre test breakfast and that the sound must not be used if violent retching be induced nor shortly after a hemorrhage. If vomiting be present a proper test meal may be taken and thus recovered, the interval being often sufficiently well observed.

Hyperchlorhydria vs. Hypochlorhydria.

General Dyspeptic Symptoms.—These are more commonly associated with varying degrees of actual pain than is usual in other ailments Because of normal or high acidity the digestion is normal or often more rapid than normal, the tongue relatively clean and the appetite often good. Indeed true anorexia is an exceptional symptom in gastric ulcer, insufficient nutrition being ordinarily dependent upon the patient's dread of the pain and distress attending the ingestion of food. **Anæmia.** The author feels that too little stress is laid by writers upon the frequency of *intractable anæmia* and regards it as one of the more important of the subordinate factors in diagnosis. It is probable that the anæmia of gastric ulcer in some cases antedates the lesion, probably invites, and, tends to perpetuate it. In others as the result of persistent or recurrent hemorrhage of varying degree, it assumes an intractable though not extreme secondary type whether antecedent or consequent. *Marked intractable anæmia of the secondary type* in young persons *associated with gastric disturbance* and normal or high acidity is suggestive, and, when accompanied by even minor degrees of persistent local pain and tenderness will justify the institution of *medical* treatment under a tentative diagnosis.

Rapid digestion.

Intractable type important.

Tentative diagnoses.

Differential Diagnosis.—*Simple hyperacidity* is commonly temporary, intermittent, dependent upon known etiologic factors and usually lacks severe pain, localized tenderness and hemorrhage.† *Gastric hyperæsthesia* not only involves the whole stomach outline but is asso-

* According to many observers it is a more constant symptom in the United States than in European clinics, the average total acidity being above 100 and the free acid above 50.

† Rare instances of vicarious menstruation with hyperacidity occur.

ciated with distinctive neurotic symptoms. *Gastric erosions* offer great difficulty in many instances but are in general to be distinguished by a more diffuse burning sensation upon the taking of food, actual pain being as a rule slight or absent, or in any event coming on immediately or within half an hour of the ingestion of food. Pressure tenderness is usually lacking and hyperacidity is the exception. The wash water of the fasting stomach contains shreds of mucous membrane, but this may occur in other conditions. Finally, it may be said that inasmuch as a case of erosion demands much the same treatment as ulcer if recognized, it is not necessary to weigh too carefully those differential points which must, the writer believes, be taken *cum grano salis*, the points of distinction being in many instances less definite and absolute than are here laid down.

May be clear.

Often obscure.

Duodenal Ulcer.—This condition, obscure and frequently overlooked, is suggested by the symptoms of ulcer attended by maximum pain to the right of the median line, with absence of blood in the vomitus or stomach washings and frequently its presence in the stools. Hyperacidity is the *exception* in duodenal ulcer, slight jaundice is not uncommon and the pain accompanying the taking of food usually becomes severe only after two or three hours. *As between ulcer of the pylorus and one of the duodenum* an absolute diagnosis is usually impossible. *Carcinoma of the stomach* presents little difficulty save in the rare subacid ulcer cases or those in which a carcinoma develops upon an old ulcer base, in which case *the symptoms of ulcer at first predominate and are gradually submerged in those of carcinoma.** The pain of cancer is ordinarily less sharp and paroxysmal, more continuous, the superficial or deep tenderness less in degree. A palpable tumor makes its appearance in a large majority of the cases and is usually right sided, solid and firm, whereas in ulcer, save in pyloric cases or where adhesion and perforation have occurred,if any tumor is felt on the wall it is thin and disc-like representing the inflamed ulcer base, and usually *left sided*. The nutrition suffers greatly in carcinoma and is associated with the progressive development of the well known cachectic appearance and morose facies. Hemorrhage is more consistently of the oozing type, appearing as "coffee ground" detritus in the vomitus and stomach washings, the location of the pain is usually to the right of the median line, because of the predominance of cancers at the pylorus

Pain.

Blood in stools.

Puzzling cases.

Usually clear.

Marked differences.

* It is evident that these are at certain periods undiagnosticable, yet repeated examinations will show the gradual and significant transition. See note, p 271.

and finally and most conclusively an analysis of the stomach contents reveals with rare exceptions an absence of free hydrochloric acid, the presence of lactic acid and in a majority of instances the Boas-Oppler bacillus which is differentiated from the mouth bacillus of a similar type by staining a mount with Gram's solution, which colors the Boas-Oppler brown and its impersonator blue.

ULCER WITH ADHESIONS.—The diagnosis of ulcer with adhesions depends largely upon the general symptoms of ulcer as stated, combined with those referred to adjacent organs and, sometimes, a sensation of dragging or pulling experienced in certain movements or attitudes. Very often a tumor may be palpated but many cases offer none but indefinite or misleading signs. **Perforation.** The symptoms of perforation are those of surgical shock succeeded by symptoms of local or general peritonitis and *occurring in cases ordinarily giving a history of ulcer.* They are essentially surgical and need not be further considered. *It is evident that gastric ulcer is not a single symptom disease, but* one that often requires a careful estimate of *probabilities*, and a thorough knowledge of all of the diagnostic factors involved. *No doubt an immense number are overlooked and go on to spontaneous healing, certainly many die of sudden hemorrhage or perforation without previous recognition.* Having these facts in mind, the author believes that wherever any reasonable probability exists that a given case is one of ulcer, the physician should not wait for more light but should at once institute appropriate medical treatment.

Dragging.

Basis of diagnosis.

Proper procedure.

Operative Cases.—The tendency to perform radical surgical operations of the major type as a primary measure in gastric ulcer is to be deplored, and many such cases are undoubtedly those of simple erosion. *Nearly every case even of true ulcer should first receive prolonged medical treatment* and the knife be reserved for the obstinate and recurrent, persistently hemorrhagic, or perforative cases, for those associated with adhesions which tend to impair the health or persistently annoy the patient, or, for actual stenosis.

A bad custom.

GASTRIC EROSIONS.—Under various conditions and influences both local and remote the gastric mucous membrane may show a surprising vulnerability, and erosions are easily produced varying from a pin's head to the size of a lentil. Various names have been given to this condition and Einhorn has described it as a separate disease, but in view of the fact that it may occur under purely nervous influences, in chronic gastritis, achylia gastrica, and probably as an early stage of gastric ulcer, it should hardly as yet be dignified to that extent.

Vulnerability of mucosa.

Symptoms.—These may closely simulate gastric ulcer with considerable or small hemorrhages and be entirely symptomless or blend with the symptom complex of the various diseases above mentioned. In those cases simulating ulcer the *pain is usually less intense*, and *comes on earlier* but may continue until the stomach is empty. *Stomach washings show tiny shreds of membrane, local tenderness is not marked* and *mucus is usually present* in the vomitus or removed contents. *Diminution of hydrochloric acid* is the rule. Occult blood in the feces and slight staining of the stomach washing are common. *Under proper treatment recovery is usually prompt.*

Occult blood.

Comment.—Differential diagnosis is important only as preventing unnecessary operations as the medical treatment of the two conditions is almost identical.

CARCINOMA.—Etiology.—*Predisposing Causes.* 50% of all cases of carcinoma in the male are gastric and age markedly affects the ratio of incidence. Such growths are rare under 30, but are always possible.* It is most frequent in males, and heredity is a factor of considerable importance. The scar of a pre-existing gastric ulcer occasionally undergoes malignant change. Traumatism seems in rare instances to be a factor and some authorities lay much stress on mental worry or strain, but it is probable that these conditions exercise little influence.

Incidence.

Causes unknown.

Morbid Anatomy.—Cancer of the stomach is usually primary, though occasionally it is secondary to cancer of the breast, uterus or other organs. The most common growth is the cylindrical celled adeno-carcinoma. Encephaloid is next in frequency, then scirrhus and colloid. Malignant growths of the stomach are frequently the source of metastases in their advanced stages. Such tumors usually develop near the orifices and most commonly at or near the pylorus (50%). In this situation they tend to cause obstruction and dilatation. Growths at the cardia (9%) are often associated with atrophy and occasionally marked esophageal distension. The stomach may be greatly displaced as well as dilatated in the case of pyloric growths and adhesions may obscure and complicate the clinical picture. Such tumors are ordinarily, however, freely movable and may in rare instances be found in almost any part of the right half of the abdomen.

Usually primary.

Types.

Sites.

Dislocation and adhesion.

* The author recalls a serious blunder due to the fact that the extreme youth of the patient made him loath to accept what should have been positive evidence of malignant growth; nevertheless, the disease is rare under the age of 30, uncommon between the ages of 30 and 40, most frequent between the ages of 40 and 70.

Symptoms.—Cases may reach an advanced stage of development without producing any recognizable symptoms save those made evident by examination of the stomach contents. Ordinarily one finds:—(a). *Dyspepsia.* Eructations, pressure, heavily coated tongue, foul taste in mouth, etc. (b). *Progressive loss of weight.* (c). *Marked progressive impairment of strength and endurance.* (d). *Pain.* Usually dull, boring and somewhat persistent. This symptom is present in 90% of all cases, is usually epigastric but may be referred, especially if adhesions exist, or if the stomach is greatly displaced and, in rare instances, may be severe and spasmodic. *Tenderness* may or may not be present *and is usually not extreme.* It is most marked in the *right* epigastrium and may be associated with tenderness in the lower dorsal region, posteriorly, or, anteriorly, at various points between the nipple and the umbilicus. (e). *Anæmia.* At first moderate, this may later become extreme, usually preserving a definite secondary type (see anæmia), but in advanced ulcerative cases showing at times extreme grades closely simulating that of atrophic gastritis or pernicious anæmia. Leucocytosis is usually present in metastasis or rapid ulceration. (f). *Fever.* 50% *of the advanced cases are accompanied by a moderate fever of the hectic type,* in rare instances associated with "chill and fever," due probably, to septic absorption from an ulcerating surface. (g). *Urinary signs.* The only important urinary findings are indicanuria, which is usually present to a marked degree, and, the diazo-reaction, as reported by the author in certain advanced cases. (h). *Cardiac weakness.* Marked cardiac weakness and edema of the lower extremities are usually terminal symptoms. (i). *Anorexia.* This is usually marked and often associated with repugnance, especially towards red meat. (j). *Nausea.* This is present in a great majority of the cases. (k). *Vomiting.* A symptom present in 90% of the pyloric cases and occurring usually one or more hours after meals. It is ordinarily occasional, but may be frequent or very rarely almost continuous, tending to rapid exhaustion and death. (l). *Hemorrhage.* In from 20 to 30% there is recognizable hemorrhage and it undoubtedly occurs in a much larger portion. In all cases of suspected cancer of the stomach, the stools should be examined as well as the vomitus and stomach contents. Bright red arterial blood in the vomitus is unusual, it being ordinarily in small dark grumous clots or "coffee ground" particles, because of the action of the digestive juices. (m). *Cachexia.* In advanced cases the skin is pale, yellow, inelastic and ill fitting because of malnutrition and loss of weight, and often shows areas of pig-

Seldom severe.

Usually moderate.

May be extreme.

Common.

Indican and diazo.

Peculiar.

Constant symptoms.

Frequent.

Stools.

Coffee grounds.

Wasting.

mentation or pigment atrophy. A curious earthy pallor is often present and is more or less characteristic, and, quite as striking is the peculiar facial expression so often encountered in these cases the patient appearing saturnine, anxious and depressed. (n). *Tumor.* A palpable tumor may be found in about 80% of the cases, may occupy almost any position and be freely movable or attached by adhesions. Osler has reported several cases in which a faulty diagnosis was originally made and every clinician can recall like instances. Unless fixed by adhesion such tumors are more or less movable in respiration. The liver should in all cases be carefully examined for the presence of metastatic nodules.

Pallor.

Location.

Source of error.

Stomach Contents.—To obtain the stomach contents one should thoroughly wash out the stomach in the evening, giving a test breakfast in the morning, and should use a Boas test meal (see page 238). The preliminary washing may show an excessive quantity of fluid or give evidence of imperfect digestion, impaired motility and pyloric obstruction (stasis).* On the other hand pyloric incontinence or duodenal stenosis may be indicated by the persistence of large quantities of bile.

General findings.

Chemical Findings.—Hydrochloric acid is seldom present and then not in any considerable quantity. It is probable that most of the cases in which free hydrochloric acid is found have their origin in an old ulcer base or scar.† The ferments are either entirely absent or greatly reduced, lactic acid is usually present and motility and absorption are greatly diminished.‡

Lactic acid.

Microscopic Findings.—The microscope may reveal fragments of growths and evidences of hemorrhage, the Boas-Oppler bacillus, and, absence or sparseness of yeast fungi or sarcinæ.

Boas-Oppler bacillus.

Differential Diagnosis.—The following facts should be borne in mind, viz., that in *chronic gastritis*, the hydrochloric acid may be diminished or absent and the ferments more or less inactive, but in this disease *lactic acid is not present, pain is absent or slight, hemorrhage does not*

Chronic gastritis.

*Substances are sometimes retained for many days as was the case with a segment of orange brought in by a patient under the impression that it was a cyst, the acid of the gastric juice having completely changed the appearance of the undigested food.

†B. Moore, Lancet, p. 1121, 1905, reports gastric subacidity or anacidity in two-thirds of all cases of cancer affecting *other* organs.

‡Estimation of the albumin contained in the normal saline solution used for washing the previously emptied and washed stomach involves too much *handling* as it is present only in *ulcerating* carcinomata or occasionally in gastric *ulcer*.

occur except in connection with cirrhosis of the liver, *emaciation* is seldom as early, progressive or extreme and, a tumor is not to be found.

Achylia gastrica.

In *achylia gastrica* all findings are negative, *hydrochloric acid, lactic acid and the ferments are absent,* there is usually no pain or hemorrhage and never tumor. *Emaciation is usually not marked or progressive,* though occasional exceptions occur.

Ulcer.

In *gastric ulcer, hydrochloric acid is usually normal or in excess, lactic acid is absent, ferments are normal, emaciation is ordinarily slight and a tumor is absent* save in the rarer instance when one may feel the inflamed base of an ulcer, a thickened pylorus or a mass of adhesions which may have resulted. *Ulcer has only pain and hemorrhage in common with carcinoma,* its hemorrhage is usually arterial, its pain more paroxysmal, directly related to the taking of food, and usually, markedly or completely relieved by emptying the stomach or by the ingestion of bicarbonate of soda. Cancer occurs in the old, ulcer more frequently in the young. *Finally, the diagnosis of cancer must rest chiefly upon the presence of lactic acid after a Boas test meal, evidence of hemorrhage, the Boas-Oppler bacillus, early and progressive loss of strength, emaciation, and, tumor.*

GASTRIC CRISES.—Lightning like attacks of agonizing, cramping, epigastric pain, associated with nausea or violent vomiting, at first of but a few minutes' duration, later persisting for hours or days, separated at first by long intervals, but progressively increasing until for a period of weeks they may occur daily, are characteristic of tabes dorsalis in most instances* and *may constitute the first symptom. The recession* is usually as abrupt as the onset, but unusual cases are encountered in which pain is absent, or, the critical termination may be replaced by slow tedious improvement. Intestinal and rectal crises of much the same type also occur. As an early symptom it is rare but important and extremely difficult to determine in the absence of tabetic symptoms because of its resemblance to many other conditions. A history of syphilis and the increasing frequency and duration of the attacks combined with their dramatic suddenness of onset and recession are valuable factors in differential diagnosis.

Tabes.

Variations.

Important data.

ARTERIO-SCLEROTIC ABDOMINAL CRISES.—Frequent attacks of severe paroxysmal pain of brief duration (15–30 minutes) induced by exertion or emotion and frequently by the recumbent position, if occurring in men past forty suggest arterio-sclerosis. The aortic region is usually tender and another type is associated with true

*Similar crises may be encountered in developed spinal paralysis, multiple sclerosis, morphinism, lead colic, etc.

angina pectoris. The attacks may show the same tendency to progress over a *period of many years* as is the case in tabetic crises but are more frequent, less sudden in onset and recession and lack the gastric disturbance. The age, the presence of arterio-sclerosis, the lack of relation to meals or constant gastric findings should suffice to exclude ulcer. Max Buch claims that the relief afforded by diuretin 3–4 gms. and tincture of strophanthus 15 to 24 min. daily is of diagnostic importance.*

SYPHILIS OF THE STOMACH.—At the period of general infection dyspeptic symptoms are not uncommon though usually indeterminate or taking the form of hemorrhagic erosions due to specific endarteritis. Gummata are by no means as uncommon as formerly supposed and clinically the syphilitic cases may resemble simple chronic gastric catarrh, ectasia, erosions, ulcer or carcinoma. Three particularly interesting cases have come under the author's observation. In one, an old gentleman of 70, there was a *palpable pyloric tumor*, stenosis with marked dilatation, extreme gastric intolerance, tenderness and emaciation. The cause was suggested by Hutchinsonian teeth in a daughter. Another was a steeple climber formerly a sailor æt 35, who presented a flat but definite and exquisitely tender plaque in the left epigastrium and extreme paroxysmal pain. The 3rd was a young man of 27 who presented the clinical symptoms of malignant pyloric stenosis. In all three specific medication promptly *relieved the pain* and resulted in complete disappearance of all symptoms. Excessive pain and tenderness with signs of carcinoma were the prominent features and in all the evidence of past syphilis was directly or indirectly adequate. Coffee ground vomitus was present in the first and third. The first and second cases were remarkable because of the palpable tumors.

Not excessively rare.

Three cases.

TUBERCULOSIS OF THE STOMACH is so rare as a primary disease as to be a clinical curiosity. Secondary lesions are common in pulmonary tuberculosis. The signs of ulcer, the presence of the tubercle bacillus and the pre-existing disease in secondary cases make the diagnosis.

CONGENITAL STENOSIS OF THE STOMACH.—This interesting condition has been reported sufficiently often by reliable observers to entitle it to a definite place in clinical medicine. Its symptoms are vomiting, constipation, dilatation, increased peristalsis

* In a case observed by the author the pain occurred both as an isolated epigastric crisis and in combination with typical angina pectoris.

and, in most instances, palpable tumor representing the thickened pylorus. In some instances a tumor has not been felt and in a recent case (McCaw and Campbell) the obstruction was apparently due to redundancy of the mucous membrane with obstructing folds. (See also p. 258.)

DISEASES OF THE INTESTINES.

LINES OF INVESTIGATION.—Careful inquiry should be made as to *antecedent diseases*, especially dysentery, typhoid fever and appendicitis and the latter often necessitates a careful cross examination covering the actual symptoms present in misnamed attacks. As in diseases of the stomach, the *habits* of the patient as regards smoking, drinking, meal hours, the *time consumed at meals*, *character of the diet*, and the *condition of the teeth* are important.

THE FECES.—The character of the stools demands attention. Their consistence, size, number, form, quantity, color and the admixture of mucus or blood are the chief features. The presence of constipation, diarrhœa or pain and the relation of the two latter to meals or a fasting period should be thoroughly gone into. In making inquiry concerning constipation it is best to ask whether the bowels move *daily without medicine*. In the case of diarrhœa one should know whether it is nocturnal or diurnal and induced or increased by mental excitement or emotion. In certain forms the loose stool invariably follows a meal. Finally it should never be forgotten that the stomach may be at the bottom of the intestinal disturbance. (Late achylia gastrica and chronic gastric catarrh, hyperchlorhydria, etc.)

Line of inquiry.

Remember stomach.

Normally the stools consist of food remnants, epithelium, salts, bacteria and traces of the digestive fluids. The color depends upon the diet and is normally brown or brownish yellow, being darker on meat diet, light yellow on milk diet. Certain drugs as iron and bismuth produce a black stool; senna, rhubarb, santonin and calomel a greenish yellow tint. In prolonged constipation and in certain forms of chronic obstruction, "goat droppings" balls, scybalæ, or hardened masses grooved by intestinal folds may be encountered or, the pipe stem or ribbon like stools suggesting but not proving organic obstruction in the lower bowel. On the other hand all grades of liquid or semi-liquid stools may be met with in the diarrhœas; such as the so-called "pea soup" stool of early typhoid or the brilliant ochre liquid of the later stage and the peculiar rice water stools of true cholera and cholera nostras.

Normal findings.

Peculiar forms.

Food Remnants.—Consist chiefly of undigested vegetable matter, never, macroscopically, of meat save in the serious intestinal diseases.

Blood from the lower bowel may be red or, if dark, is not usually coagulated; if from the stomach or upper bowel it presents a tarry appearance. Mucus may appear as a membrane, or as an envelope covering the stools, or mixed with the feces, the significance being that of intestinal catarrh. Pus if present proves a lesion of the lower bowel and fragments of tumor may yield information of value. Intestinal parasites are considered on page 494. Blood, mucus and pus.

Collecting the Specimen.—The simplest method consists in the use of one of the covered receptacles which can be placed within and rest upon the basin of a water closet or directly under the patient lying in bed. These can be procured with a removable wire screen which is useful in washing the stools for concretions. As an emergency measure any receptacle may be used and for a sieve one may employ ordinary mosquito netting fastened to an extemporized hoop. Sieves.

Microscopic Examination.—The portion to be examined is prepared by adding a small amount of physiologic salt solution and a few drops of 1% formaline solution. Normally the appearance varies with the diet. In ordinary mixed diet there are various vegetable cells usually characteristic, starch granules are ordinarily absent or if present are readily demonstrated by a drop of Lugol's solution. Their presence is pathologic. Meat fibres in small numbers may be recognized as may elastic and connective tissue fibres. Oxalate of lime and fatty acid, calcium carbonate and calcium and ammonio-magnesium phosphate may be present in characteristic crystals. The Charcot-Leyden crystals occur in pulmonary tuberculosis, typhoid fever, dysentery, and, with intestinal parasites. Ruby red or orange yellow rhombic plates of hematoidin, or its amorphous forms, may be present in hemorrhage or severe catarrhs; fat may also be present as characteristic highly refracting globules and both fat and fatty acid crystals are increased in diseases of the liver or pancreas, and, in acute enteritis. Technique. Findings.

Blood cells may be present if from the lower bowel and pus corpuscles if there be an ulcerative process or discharging abscess. Tumor fragments, mucin threads and the large variety of bacteria, most important of which pathologically is the bacterium coli commune and the tubercle bacillus.

Schmidt's Method.—A formal and extended examination of the feces is ordinarily too exacting and time consuming for the general

practitioner but one of the simplest methods is here given for the sake of completeness.

Simple.

Test Diet.—Schmidt suggests the following diet (as containing constant and sufficient calories, simple, and always obtainable), which has been slightly modified by many different observers, but represents the basis of all "normal" or test diets: 1.5 litres milk, 100 gm. zweiback, 2 eggs, 50 gm. butter, 125 gm. very rare or raw beef, 190 gm. potatoes, and gruel from 60 gm. of oatmeal, and, 20 gm. sugar, or, as used by Steele:—*Breakfast:* 2 eggs, $\frac{1}{3}$ of the amount of toast and butter, 2 glasses of milk, oatmeal with milk and sugar. *Dinner.* The steak and potatoes, $\frac{1}{3}$ of the amount of toast and butter, $1\frac{1}{2}$ glasses milk. *Supper.* 2 glasses of milk, remainder of toast and butter, 1 or 2 eggs if desired.

Method.

This need be given for only two or three days, a capsule (0.3) carmine being given with the first meal. The appearance of the dye in the stool initiates the examination and serves to measure the time of passage. Schmidt than proceeds to determine consistence, color and odor and reduces the feces to a liquid form in a mortar with distilled water. The ordinary blacked plate or a Petri dish placed over a black background then facilitates the examination. Normally

Macroscopic findings.

there should be nothing except the indigestible oatmeal hulls and sago-like particles of potato. Pathologically one finds:—(a). Mucus appearing as glassy flakes sometimes stained yellow. (b). Remnants of potato the resemblance of which to mucus may require the microscope to differentiate. (c). Remnants of muscle fibre indicating impaired intestinal ferments, absence of the enzyme (enterokinase) or abnormal motility. (d). Crystals of magnesium and ammonium phosphates indicating fermentation. (e). Connective tissue remnants differentiated from mucus by their toughness. (f). Pus or blood. (g). Parasites. (h). Concretions. (i). Foreign bodies.

Microscopic findings.

The microscopic examination merely confirms and somewhat amplifies these findings. It is recommended that three mounts be made. (1). A drop of the prepared material. (2). Same + acetic acid. (3). Same as No. 1 + a drop of dilute Lugol's sol. (iodine 1., potas, iodide 2, water 50). No. 2 is heated to boiling before covering and upon cooling will show fatty acid crystals set free by the acetic acid; if reheated these will form droplets. No. 3 shows the violet blue reaction of potato cells and certain spores.

Chemical findings.

The chemical examination consists of 3 routine tests:—(1). The reaction obtained by dropping the prepared material into a few c.c. of dilute aqueous litmus sol. (2). The sublimate test. (Equal parts of

the prepared material and a sol. of mercuric chloride (25%) should strike a pinkish red color indicating hydrobilirubin.) Unchanged bile pigment yields a green color and is pathologic. (3). The fermentation test.—5 c.c. of formed feces, or its equivalent if liquid, are prepared with sterile water and poured into the bottle of Strasburger's instrument or better the simple and easily cleaned modification of Steele which can be made by any one.* Fermentation.

The tube surmounting the bottle it filled with water, the parallel tube remaining empty; if kept at blood heat for 24 hours gas will rise and displace the water in the first tube forcing it into the outer parallel tube whose air finds an outlet through the longer inner tube; thus the amount of water displaced is a measure of the amount of gas formed. $\frac{1}{3}$ displacement is distinctly pathological. Albuminous putrefaction is indicated by a foul smell and alkaline reaction, carbohydrate fermentation by acidity.

Inferences from the Tests.—*Color.* A pink color reaction (test 2) is normal, the green indicates increased motility. Absence of color a fat stool or absence of bile. *Meat remnants.* Whether muscle or connective tissue and even if microscopic, an excess of either is pathologic. In the case of connective tissue, deficient gastric digestion is indicated or excessive gastric motility. In the case of the muscle the digestive trouble is probably intestinal. An excess of fat merely indicates deficient digestion of that material but suggests hepatic pancreatic or intestinal disease; fermentation means either poor starch digestion or intestinal disturbance. Albumin fermentation, either deficient gastric or intestinal digestion or increased motility. As a matter of fact it may be stated with regret that up to the present time the amount of information gained is too slight to compensate the busy practitioner for even the small amount of time necessarily consumed. Nothing but constant practice will suffice to establish proper standards representing the normal and the abnormal. And finally, proteid fermentation is more easily estimated by testing for indoxyl.

Concretions.—*Enteroliths.* Enteroliths originate usually in the small

*The materials needed are—a large mouthed medium sized bottle carrying a rubber cork, perforated by a glass tube. Two test-tubes are prepared each having a rubber cork with two perforations. Both are inverted and connected by a U tube the extremities of which project slightly above the inner surface of the cork, one is then pressed down over the vertically projecting tube of the bottle and holds the other tube parallel with it by virtue of the connecting U tube. Test-tube No. 2 is fitted with a small glass tube which goes nearly to the top and projects below.

intestines, are small, light in color and ordinarily of no importance, consisting chiefly of magnesia and lime. *Coproliths.* These sausage shaped bodies are most frequent in the appendix, cæcum, sacculations and rectum, rarely they attain considerable size and may cause obstruction. *Pancreatic calculi.* These may be faceted or are, usually, rough and friable they contain no bile pigment and cholesterin and are soluble in chloroform. *Biliary calculi.* These occur as well known faceted bodies, or, if in the form of sand, may require chemical tests for their identification.*

HEMORRHOIDS (piles).—This common condition, rare in children, most frequent in men, consists of a diffuse or circumscribed varicosity of the hemorrhoidal veins, either in the lower portion of the rectum (submucous) or at the anal margin (subcutaneous). The condition is induced by constipation, continuous standing or sitting (i.e. sedentary occupations), the habitual use of cathartics, pregnancy, the pressure of tumors, or obstructed portal circulation from whatever cause and is favored by the lack of valves in the hemorrhoidal veins. *They are frequently a source of hemorrhage which in turn may be the real cause of an obscure anæmia, often of a most severe type.* Furthermore the frequency of the lesion may lead to the too ready acceptance of a patient's diagnosis and a failure to recognize the existence of rectal fissure, foreign body, malignant growth, tuberculous or syphilitic ulceration or fistula. A rectal examination is too often neglected though its disagreeable features are in part removed by the use of the rubber glove or finger shield. Hemorrhoids are easily recognized if superficial, the patient being asked to strain and bear down, when the reddish blue nodules will become evident. Skin tags and condylomata are common but easily differentiated. Internal hemorrhoids may require the use of the speculum, though usually recognized by digital examination. Retained fecal matter has a characteristic feel and malignant growths are peculiarly indurated and firm. In any rectal examination the tonus, i.e. resistance offered by, and grasp of the anal sphincter should

Site.

Etiology.

Source of error.

Appearance.

Fecal masses.

Tonus.

* *Test.* Powder the stone or gravel, add 20 c.c. of ether, mix thoroughly, filter, and evaporate filtrate, divide residue in 3 portions. No. 1 is dissolved in hot alcohol, allowed to evaporate without heat and the residue examined for the rhomboid crystals of cholesterin. No. 2 is treated with HCl and a trace of ferric chloride; a blue color on evaporation indicates cholesterin. No. 3 is placed on a slide, treated with a drop of concentrated sulphuric acid and covered. The cholesterin crystals show carmine margins. Another portion may be treated with dilute HCl, heated, cooled and treated with chloroform; to the chloroform extract Gmelin's test for bile pigment is applied.

be noted. It is markedly relaxed in certain organic nervous diseases and in obstruction of the rectum and sigmoid flexure.

ENTERITIS.—In the diarrhœal diseases one must distinguish those affecting the small intestine alone from those involving the colon exclusively or in part. The latter, more severe and likely to be attended by serious complications, are described under dysentery and entero-colitis nearly all forms of *enteritis* are trivial and temporary in the case of the adult but may assume considerable importance in the case of infants and young children, occurring usually under conditions of improper sanitation or still more frequently in *artificially* or badly fed infants. Bowel disturbances in breast fed infants are rare and usually temporary and trivial unless secondary to some grave primary condition. All varieties are prone to appear during hot weather, particularly when extreme variations in temperature and humidity are present, and reach their maximum in prevalence and mortality in July and August. Milk and water are probably the chief vehicles of contagion but the demonstration of a specific bacillus for the ordinary diarrhœal diseases must await further investigation inasmuch as a large variety of organisms are found, many of which are quite competent in themselves to produce decided symptoms or to act with and intensify the virulence of others.*

Enteritis vs. Colitis.

Clinical Varieties.—We recognize (1). **Acute Intestinal Indigestion.** (2). **Acute Fermentative Diarrhœa.** (3). **Cholera Infantum.**

Morbid Anatomy.—The pathologic changes in enteritis vary from a mere congestion or catarrhal inflammation of the mucosa with slight infiltration of the submucosa and enlargement of the lymph follicles, to actual ulceration. Ulcerated areas are ordinarily limited, originating at the follicle but extending by the coalescence of contiguous ulcers. In combined lesions of the upper and lower bowel one may of course have all the changes described under dysentery. In the more severe types, particularly the combined form, broncho-pneumonia is common and the viscera present evidence of severe toxæmia.

Variable severity.

Acute Intestinal Indigestion.—Colicky pain, tympanites, and diarrhœa may come on suddenly, immediately or from several hours to a day after some dietetic indiscretion or, without apparent cause;† the fever is usually moderate and of brief duration, the pulse

A common ailment.

* Much interest attaches to a bacillus recently discovered by Duval and Bassett which closely resembles Shiga's bacillus of dysentery and has been found in a large number of cases of the summer diarrhœas of children.

† These attacks are frequently of *gastric origin.*

Stools.

rate is increased and in the child may be extremely rapid. The stools are at first fecal, then watery and contain mucus and food particles imperfectly digested; such an attack is ordinarily cut short by the administration of some appropriate cathartic, such as castor oil.

Severer type.

Acute Fermentative Diarrhœa.—This is characterized by *more marked symptoms* throughout and may come on suddenly or succeed a milder attack of acute intestinal indigestion. Vomiting is usually present, the fever may reach 105° F., the pulse is rapid, nervous symptoms are pronounced, exhaustion marked and, in children, convulsions are not uncommon at the onset.

Stools.

The stools are more frequent than in the simple form, rapidly become watery, are green in color and contain much mucus. The disease is ordinarily of brief duration and favorable termination, but may prove the commencement of an ileo colitis and may terminate fatally by coma, exhaustion or broncho-pneumonia.

Reflects primary disease.

Chronic Enteritis.—Hardly justifies a separate description, being characterized by intermittent or persistent symptoms of a mild sort which mirror the acute attacks. If long continued it impairs strength and nutrition and it may be associated with mild daily diarrhœa, mere unformed stools, or, obstinate or alternating constipation.

CHOLERA INFANTUM.—This ailment, remarkably fatal and prevalent in the large cities amongst the poor, may affect the children of any age and is both sporadic and endemic. The onset is usually

High mortality.

sudden, with high temperature.* Vomiting is usually present, and the stools at first fetid may reach 20 or more daily and rapidly become

Violent choleraic symptoms.

watery, light yellow or greenish, then colorless and odorless. There is marked thirst, the urine is scant and often albuminous and the disease usually terminates fatally, often with delirium, stupor or coma and perhaps convulsions. Marked tympanites is not present nor is the abdomen tender but there is evidence of profound collapse, the features being pinched, the skin ashy gray and the surface usually cold even though rectal temperature may be high. *Ileo-colitis* is predominatingly a dysentery and will be described under that heading; the small intestines seem to be primarily involved, but symptoms very soon become distinctly dysenteric.

DYSENTERY.—Definition.—A term applied to a group of diseases characterized by acute or chronic inflammation and ulceration of the lower bowel, diarrhœal stools, containing blood and mucus, and, associated with colicky pain and tenesmus.

*This must be taken by rectum or vagina because of low surface temperature.

Etiology.—The disease may be primary or secondary, acute or chronic. The secondary cases depend upon a primary cause, such as tuberculosis, syphilis or Bright's disease. The primary cases fall under three heads:—(1). *Amœbic dysentery.* (2). *Acute specific dysentery* (*tropical dysentery*). (3). *Acute catarrhal dysentery* (*acute ileo-colitis*).

Amœbic Dysentery.—The amœba dysenteriæ was first described by Lambl and Losch in 1859 and first identified in hepatic abscess by Kartulis, but to Osler's description (1890) and careful work we owe most of our knowledge of its importance as an etiologic factor. It is from 15 to 20 μ in diameter, i.e. about twice the diameter of the red blood cell, contains a nucleus and one or two vacuoles surrounded by an inner granular zone and a clear outer zone. It causes from $\frac{1}{3}$–$\frac{1}{2}$ of all cases of tropical dysentery and if a warm slide is used for examination the amœbæ may be readily identified by their structure and movement and are usually contained in the flakes of mucus or pus of the fecal discharges or may be obtained from abscesses or stained in situ in tissues.

Osler's work.

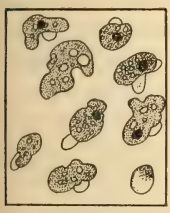


Fig. 120.
Amœba Coli.
(After Braun.)

Amœbæ readily recognized

Acute Specific Dysentery (acute tropical dysentery).—Practically all tropical dysenteries not due to the amœba are caused by Shiga's bacillus which produces the disease in animals by inoculation and is agglutinated by the blood serum of affected patients.

Acute Catarrhal Dysentery (acute ileo-colitis), (follicular dysentery).—This is the ordinary well known form of dysentery encountered in the temperate zone. No specific germs have been isolated but a large variety have been described as associated with the process.

Diphtheritic Dysentery.—This is characterized by diphtheritic inflammation with extensive infiltration and formation of sloughs. It is often secondary to exhausting chronic diseases such as chronic nephritis, heart disease and malaria.

Symptomatology.—This is essentially the same for all varieties though varying greatly in degree. Any form may become chronic, tropical forms are both endemic and epidemic, and for their development all forms of primary dysentery elect hot weather. It will be noted that the symptoms are precisely those that might be expected to follow inflammation of the lower bowel *plus* widely varying degrees of toxæmia. The *onset is sudden and violent* in all tropical forms, being often associated with chill, whereas the milder variety is often preceded by an initial diarrhœa. Abdominal distress and fever are

General type.

Violent onset.

likely to be high during the active stage of all forms and febrile phenomena are marked. *Essential symptoms. Purging, marked tenesmus, severe colicky pains, constant desire to go to stool, excessive thirst, great prostration, and, tenderness of varying degree over the descending colon are symptoms common to all forms. The stools.* These contain mucus, blood and scybala, and in the severer forms pus and sloughs. The number of stools varies greatly, but may reach 100 or more in the 24 hours. They tend to become fetid and sanious in severe and unrelieved cases. The exhaustion is great yet vomiting and gastric irritation are often absent. Emaciation is of course rapid and in severe cases extreme. Ileo-colitis is usually preceded by a short period of ordinary diarrhœal stools and it must be remembered that in children mucus and blood may appear in any severe diarrhœa.

Emaciation and exhaustion.

Chronic Dysentery.—Chronic indigestion, recurrent dysenteric attacks of varying degrees of severity and marked emaciation are the chief features in chronic cases. Many of these have been found to be due to the persistence of a malaria in soldiers returning from the tropics, but any form may become chronic and the irritability of the lower intestinal tract may last for a few weeks or months or even for many years.

Often malarial.

Differential Diagnosis.—The peculiar character of the diarrhœa with its bloody stools and *marked tenesmus* serves to distinguish the acute form; amœbæ should be searched for in suspicious tropical dysentery and the specific agglutination reaction sought. *Typhoid fever* is readily differentiated by its *gradual onset, diazo-reaction, rose spots, temperature curve and finally and conclusively by the agglutination test* with the typhoid germ. As it ordinarily wholly lacks the characteristic dysenteric phenomena confusion can seldom arise. *Tuberculous* or malignant ulcers and *foreign bodies* in the rectum may cause attacks more or less closely simulating chronic dysentery, but the associated lesion and the results of local and microscopic examination will ordinarily distinguish them.

Usually easy.

Mortality and General Comment.—The death rate is of course highest in the severe tropical forms and dysentery of the temperate zone is seldom fatal except in children and old people, or, persons exhausted by disease. On the other hand the total mortality in chronic dysentery under conditions of poor food, overcrowding and general insanitation may be very heavy. The United States government is still paying a large number of persons for disability attributed to chronic dysentery of the Civil war and the *deaths* during that four-year period reached

an enormous total. The mortality in Japan for the decade following the first introduction of the disease reached 247,000.

Membranous Enteritis (membranous diarrhœa, mucous colic).—**Definition.**—An affection characterized by the presence of mucus, in quantity, in the feces. **Etiology.**—More common in women than in men, this affection seems largely dependent upon the element of hysteria and neurasthenia, but is a relatively rare disease, most frequent in connection with an enteroptosis or achylia gastrica though met with independently of those affections. **Symptoms.**—Following an attack of obstinate constipation, violent, colicky pains occur associated oftentimes with diarrhœa, perhaps of a dysenteric type, and more or less decided dyspeptic symptoms. Mucous masses are discharged which are usually grayish white, ribbon like or membranous, rarely, forming complete molds of the intestinal canal (tubular form). Their nature may be proven by Pariser's method.*

Neurasthenic women chiefly.

Mucus.

MISCELLANEOUS INTESTINAL NEUROSES.—**Rectal spasm** is almost invariably secondary to inflammatory or ulcerative processes either of the rectum itself, the colon, the anus or neighboring organs, rarely it is supposed to be primary. Defecation is intensely painful, the anus is sensitive and resistant, an anæsthetic being required for an examination. **Peristaltic unrest** associated with borborygmi may be primary or secondary. The former is a pure neurosis, the latter may be associated with various gastric disturbances or with actual obstruction. The condition is usually accompanied by constipation and in its primary and severe form by hysteria, neurasthenia or hypochondria. It is as a rule trivial and unimportant. **Meteorism.**—Gaseous distension is a mere symptom too well known to require extensive description and results chiefly from the fermentation of carbohydrates or proteids, frequently associated with an obstruction, usually temporary and purely neurotic, but is also an important manifestation in true obstruction and peritonitis. It may prove a troublesome and dangerous condition in the typhoid state or any virulent infection and is particularly dangerous in pneumonia and chronic incompensated disease of the heart. **Enteralgia** has more basis in fact than gastralgia being descriptive of ordinary colicky pain of intestinal origin whether it be primary or secondary.

Sometimes serious.

Hypogastric Neuralgia.—This term has been applied to a painful condition localized in the epigastrium and lower part of the back and

*They are treated with sublimate alcohol and then yield a green color with Ehrlich's triacid stain in contradistinction to fibrin which stains red.

associated with sense of pressure in the rectum, bladder, uterus and vagina. It is purely a symptom, undoubtedly dependent upon uterine, ovarian, hemorrhoidal and other similar conditions. **Hyperæsthesia, Paræsthesia, Anæsthesia.**—The intestines share with other portions of the body these symptoms of hysteria or neurasthenia and they not infrequently take the form of aura in epilepsy. They are important only to the possessor of them save in actual disease of the brain and cord when rectal anæsthesia may be associated with sphincter paralysis, or, in the cases where the anæsthesia leads to a serious accumulation of fecal masses. **Intestinal neurasthenia** is given a place by most authors but it is doubtful whether the condition should be so dignified. The term really applies to the neurasthenic state with predominance of abdominal symptoms. **Paralysis of the Intestines.**—This has already been referred to on page 65. The condition may be a very serious one and presents many of the symptoms of obstruction. It is associated with direct abdominal injury, laparotomy, chronic inflammation and ulcerative diseases of the intestinal tract, with profound hysteria, melancholia, hypochondria or actual organic disease of the brain and cord and is frequently observed in prolonged and *massive* fecal impaction.

CHRONIC INTESTINAL OBSTRUCTION.—Acute obstruction has been dealt with on page 64. Chronic obstruction may be due to the same factors and one is chiefly concerned with its recognition.

Often obscure.

Symptoms.—These are extremely variable and often obscure. The history of previous ailments is important as is the history of constipation, diarrhœa or symptoms suggesting possible ulceration or appendicitis. Little stress should be laid upon the pipe-stem or tape like conformation of the stools as these may be met with in spastic constipation or anal spasm. The gradual onset of the symptoms is characteristic. At first hardly noticeable, they may become severe and troublesome and always depend greatly upon the site of the obstruction. *Duodenal stenosis* produces symptoms strongly resembling pyloric obstruction and is frequently associated with the vomiting of bile in quantity thus indicating a stricture below the papilla of Vater. Any marked obstruction in the *small intestine* will be accompanied by a great increase of indoxyl, and the more remote it is from the stomach the more will the colicky pains and constipation predominate over the gastric symptoms, nausea and vomiting. If in the *lower ileum or colon* there may be no gastric symptoms. Local distension of the abdomen or visible peris-

Gradual onset.

Regional variations.

talsis may give a clue to the site and gurgling and bubbling sounds may be audible and palpable with or without the pressure of the hand. It may be possible to recognize the large peristaltic waves of the colon as compared with those of the lesser intestinal coils, but it is evident that this sign must often fail.

Chronic intussusception is usually associated with palpable tumor, bloody stools and tenesmus as in the acute form, its most frequent site being at the ileo-cæcal valve. Old appendiceal adhesions may produce chronic obstruction in the same region but lack all distinctive symptoms of intussusception. In this connection it may be said that following appendicitis, adhesions may occur about the cæcum or the ascending portion of the colon producing obstinate constipation and frequent attacks of more or less extreme pain though the appendix is found at operation practically normal and free.*

Tumor and bloody stools.

Adhesions after appendicitis.

CONSTIPATION.—This common and often troublesome symptom may be caused (a) *By the character of the food taken*, being more common in those taking an exclusive meat diet than in vegetarians. (b). *Disturbances of innervation*, more frequently than from any other cause. It is seen in hysterical neurasthenia, brain injuries, simple neglect or lack of proper habit, injuries and disease processes affecting the peritoneum, lead poisoning, nervous dyspepsia, hyperchlorhydria, etc., etc. (c). *Mechanical causes*, amongst which are weak abdominal musculature, strictures, growths and the recumbent posture. (d). *Reflex causes* and direct irritation as in fissure of the anus, hemorrhoids, rectal ulcer, prostatitis, and displacement of the uterus or ovaries. (e). *Fever* and other conditions associated with profound toxæmia, diminished secretion or excessive abstraction of fluids. (f). *Chronic disease of the stomach, colon or small intestine.*

Etiology.

Clinical Divisions.—One distinguishes an *atonic and a spastic form*, the former representing either a weak intestinal musculature or impairment of its nervous mechanism. This is seen in chronic venous congestion (chronic heart disease, hepatic cirrhosis, etc.) or in states of profound general debility, neurasthenia, hysteria or certain organic nervous ailments.

Atonic.

Spastic constipation is characterized by permanent increased tonus of the intestines and the rectal segment. The spasm may be of variable duration and involve one or many intestinal segments in varying degree. If the whole small intestine is affected a scaphoid abdomen is produced as is seen in spinal meningitis

Spastic.

* The author has had several of these cases operated upon with excellent results.

Stools.

Colic.

or in certain irritative cerebral lesions. More frequently the colon is involved and yields no symptoms on inspection save that the stools resemble round hard balls often like goat droppings or the pipe-stem or tape like forms. One somewhat characteristic symptom of spastic constipation is intermittent pain in the left lower abdominal segment relieved by stool or enema. This at times is very severe and constitutes a type of colon colic.

Etiology.

Hospital cases.

Various sequelæ.

FECAL ACCUMULATION is most common in persons suffering from profound toxæmia in the acute infectious diseases such as typhoid, in the insane, or, in profoundly hysteric or neurasthenic individuals. Careless physicians, house officers or nurses are pretty sure to encounter them. They are sometimes associated with profound auto-intoxication, partial or complete intestinal paralysis and acute gastric dilatation. Such masses frequently produce neuralgic symptoms or even a sciatica and, if the condition be of long duration, the utmost difficulty will be encountered in attempting to relieve it by enema or by physic. Rectal obstruction is easily diagnosed by the finger and speculum. It may take the form of irregularity, greatly diminished frequency, or, a mere diminution in quantity, the fecal passages being chronically insufficient.

Readily detected.

In fecal impaction and indeed in almost all forms of constipation palpation of the rectum or even the relaxed abdominal wall is sufficient for the detection of fecal masses. If actual obstruction occurs from this cause ileus is closely simulated, otherwise the symptoms are too well known to require description.

Obscure.

DUODENAL ULCER.—Reference has been made to this condition under gastric ulcer from which it can rarely be distinguished. Its etiology is unknown save in the case of extensive superficial burns of the skin which frequently produce it. It is less frequent than gastric ulcer in the ratio of 1–12 or 15, occurs ordinarily in older persons (between 30 and 60) and occasionally affects children, even in infancy.

Site.

Its most frequent site is the upper ascending or horizontal portion of the duodenum, immediately behind the pyloric fold. Its relation to the diverticulum of Vater when it occurs in the descending duodenum makes it likely to involve the pancreatic and biliary ducts. It may be single or multiple and may cause a localized peritonitis by perforation.

Symptoms.—These closely resemble ulcer of the pylorus though the *gastric symptoms are usually markedly less. Hemorrhage* occurs in 25 or 30%, *but blood only occasionally appears in the vomitus.* Symptoms of perforation have already been described on page 263. *Symptoms*

of icterus occasionally occur and help the diagnosis and the *pains do not radiate to the back and are usually distinctly right-sided.*

Prognosis.—Complete recovery is rare, relapse is common, perforation more frequent than in gastric ulcer. The hemorrhage is often severe, occasionally fatal and malignant growths may develop upon the ulcer base.

THROMBOSIS AND EMBOLISM.—This rare condition results usually from a clot in the left auricle in cases of mitral disease, thrombosis being the common form and the *superior mesenteric* the almost invariable site. The condition can seldom be recognized antemortem. Septic emboli carried into the *smaller branches of the intestinal arteries* from an endarteritic or septic focus may cause ulcer and produce severe colicky pain, localized tenderness and bloody diarrhœa. Embolism of the inferior mesenteric artery is very rare, the symptoms not lending themselves to exact diagnosis.

Rare and obscure.

TUBERCULOSIS OF THE INTESTINES.—This is rarely primary but a common secondary manifestation, occurring in a large percentage of the advanced pulmonary cases. It affects chiefly the lower portion of the ileum and may extend downward even to the rectum or to a variable extent upward. The ulcers are irregular in shape with unclean bases and bright red margins which are usually undermined or overlapping. They readily form adhesions and hence seldom perforate. Owing to their tendency to encircle the intestine they may in rare instances heal and produce cicatricial stenosis. Diagnosis is evident from the pus, blood and tubercle bacilli in the stools, associated with localized pain and tenderness, and, usually, a pre-existent known tuberculosis.

Primary, rare.

Ulcers.

Secondary, common.

SYPHILIS OF THE INTESTINES.—Syphilitic ulcers formed by the softening of gummata occur, congenitally, in the small intestines, but in the acquired form, chiefly in the colon, rectum and anus. They present a bacon like appearance with well defined margins and an indurated base. Ulcers are occasionally observed in secondary syphilis and a form of ulceration of indefinite causation termed "toxic" occurs occasionally in connection with the cachexias of leukæmia, scurvy, nephritis and the like.

Ulcers.

Intestinal Parasites.—See page 494.

ENTEROPTOSIS (Glenard's disease).—*This syndrome represents a ptosis involving the stomach, intestines, kidneys, liver and spleen.* In one type characterized by absence of subjective symptoms, the condition is associated with *extreme* relaxation of the abdominal wall, either from repeated pregnancies or recurring ascites, which

Two types.

Symptomless form.

Its opposite.

often involves separation of the recti. In the other, the same condition in lesser degree is associated with marked neurasthenia and the victims are usually young, thin females. **Symptoms.**—*The first group is one of outward signs, the second of subjective symptoms*, and the absence of complaint in the former is striking in contrast with the apparent disturbance of normal relations, the abdomen being relaxed and usually thin, the stomach if inflated being plainly visible and greatly displaced, while intestinal peristalsis and to a slight degree the gastric waves may be evident. If gastric dilatation be present it is likely to be that of atony, the peristalsis lacking the vigor of that seen in pyloric stenosis with ectasia. Palpation shows displacement of the kidney (see movable kidney, page 365), sometimes a less marked displacement of the liver and, occasionally, decided mobility of the spleen. The colon is so prolapsed as to form a more or less perfect V and *may be felt as a cord* crossing the abdomen at or below the navel. In the second group there are symptoms of nervous dyspepsia, dragging pains usually lumbar, *but often sacro-iliac*, subjective epigastric or umbilical throbbing and, in short, the combined phenomena of nervous dyspepsia and more or less pronounced neurasthenia. Not only do we frequently find the first type of cases lacking in symptoms but even those of the second type may develop only after an exhausting illness, some profound emotional disturbance, or the disclosure of the diagnosis by the physician.

Objective vs. Subjective.

Ptosis.

Colon.

Pain.

Latent cases.

APPENDICITIS.—*The remarkable prevalence of appendicitis as a disease is due merely to its substitution under correct pathology for the old term peritonitis which formerly covered its secondary and most fatal sequence and to a better knowledge of the sources of abdominal pain.* Nowadays, aside from traumatism and perforating ulcer of the stomach and duodenum, acute general peritonitis is rare in the male save as a result of appendicitis, while, in the female, appendicitis is overwhelmingly predominant though the pelvic structures share the dubious honor

Mortality.

Prevalence.—Appendicitis causes upwards of 2% of all deaths in the United States, a higher mortality than is shown by the statistics of foreign countries. **Etiology.**—The disease is more prevalent in the young, being most frequent under the age of 30 and comparatively rare as a primary lesion in persons of middle age. It is *slightly* more frequent in the male than in the female and in the latter is frequently *combined* with inflammation of the pelvic viscera.* An indiscreet

Age.

Sex.

* Dr. A. McLaren and other surgeons of wide experience believe this male predominance a myth.

diet no doubt plays a part, particularly in recurrent cases, and occupation is a factor, especially that involving hard muscular work or severe strains such as lifting. Occupation.

Symptoms.—Acute appendicitis yields symptoms varying with the nature and extent of the inflammatory process, but all showing certain well defined characteristics. The *subacute* and *acute catarrhal form* in which the local changes are slight *may yield no definite symptoms* or signs, *or, disproportionately and misleadingly severe ones.* Obscure cases. The *acute diffuse form* is associated with an active inflammation and infiltration, often with erosions of the mucosa, and produces usually but not always marked symptoms. Purulent and gangrenous appendicitis are advanced forms of the same primary lesions. Frank cases. *Abdominal pain, fever and localized tenderness* are the essential features of all acute forms. Essential symptoms. *Pain* of sudden onset *and a tendency to localize itself* within 48 hours, usually earlier, at a point representing the intersection of the outer edge of the right rectus with a line drawn from the anterior superior iliac spine to the umbilicus (McBurney's point) and the associated *localized tenderness* really make the diagnosis. McBurney's point. *Fever.* Without fever a positive diagnosis of acute appendicitis is difficult, yet cases occur in which local and even general peritonitis are present without fever. *Vomiting* and *constipation* are usually marked features, *children may show diarrhœa.* **Physical Signs.**—Early in the disease Lacking early. there are none of importance save the evident pain and distress and fever. *Palpation* may show *increased resistance* (muscular rigidity) with or without tenderness in the region of McBurney's point, *but in many cases this symptom is delayed for 24 hours or more.* Doubtful *early* cases justify McMonagle's manœuvre, i.e. fixation of the knee in extension and asking patient to attempt flexion of the thigh on abdomen against resistance, while the unoccupied hand palpates the abdomen. Cutaneous hyperæsthesia of the 11th dorsal nerve may be present. The relaxation of muscular rigidity under anæsthesia in perforative cases is said to occur last over the exact site of the perforation. *Rectal or vaginal examination may yield important information in obscure cases* Important. *and should never be omitted. Percussion* is of no special value and *auscultation* is negative. As the disease advances exquisite local tenderness develops, there is a tendency for the patient to draw up the Tenderness. leg of the affected side, and in certain cases palpation may reveal an actual induration, sometimes in the cæcal region, more often just above Poupart's ligament. Bladder irritability is common and, in the female, involvement of the appendages by inflammatory adhesions is frequent.

Shock and collapse.

Perforation and General Peritonitis.—Temperature may be low or absent, the pulse slow, the pain may disappear and the patient believe himself better, but soon the facies assumes the Hippocratic cast, the abdomen is distended, motionless and strikingly resistant. Both knees are drawn up, the pulse becomes rapid, wiry, and small, the tongue dry. *It should never be forgotten that in the earlier stages of appendicitis the pain and tenderness may be general, and that in perforation and general peritonitis the patient's sensations and the temperature may be most misleading.*

Misleading signs.

Sudden pain.

Differential Diagnosis of Acute Appendicitis.—Any attack of acute abdominal pain attended by fever should at once *suggest* appendicitis as the cause, the suddenness of the onset being an important though not absolute distinction as regards typhoid, for which it has been many times mistaken. *Renal colic* may prove deceptive in two forms, *first*, the so-called Dietl's crises which usually lack the ultimate localization of appendicitis. *Secondly*, the passage of calculi through the ureter and especially such as are temporarily lodged in its lowest and narrowest portion. The recognition of renal colic should offer no difficulty by reason of the localized original pain over the kidney and its distribution. Furthermore, even the ureteral cases are usually made clear if the urine be examined *as it always should be before any operation is undertaken.* Several times in the author's experience such patients have narrowly escaped the knife. *Gall stone colic* is usually definitely localized both as regards points of tenderness and referred pain and *pelvic peritonitis* presents bilateral rigidity, hypogastric pain and localizing symptoms per vaginam. Nevertheless *acute tubal or ovarian inflammation* or a pelvic situation of an inflamed appendix *may complicate the diagnosis*, and a local examination is always necessary in women. *Strangulation* and intussusception differ from appendicitis in the fecal vomiting of the former and bloody stools of the latter.

Examine urine.

Pelvic disease.

Wide variations.

Prognosis.—Mild catarrhal cases recover without operation after a few days' illness, those going on to suppuration may result in a general peritonitis, gangrene, or perforation attended with extensive purulent inflammation in remote regions, one of which is the perirenal tissue. Certain of the acute suppurative cases rupture into the bowel, draining the abscess cavity, and may show a remarkable freedom from recurrence. The question of surgical interference in appendicitis is a difficult and important one. *Surgical consultation should be the invariable rule, and surgical judgment should ordinarily be accepted.* No matter how mild the case, a second attack usually calls for operative interfer-

Bowel drainage.

Recurrence.

ence and in those who live in, or whose duties take them to remote regions, a primary or interval operation should be the invariable rule.*

CHRONIC APPENDICITIS.—As physical signs may be wholly lacking and the symptomatology suggestive of disease of other organs, *the diagnosis usually depends upon a definite history of antecedent acute, subacute or recurrent appendicitis, which may masquerade under the title of "typhoid fever," "inflammation of the bowels," "gall stones," "renal colic," "gastralgia"* or *"ovarian trouble."* Occasionally there are enlarged and thickened appendices or old inflammatory masses that may be palpated. Quite frequently there are mild but definite localizing symptoms and slight fever (99.5 F) at the time of examination. *Suggestive symptoms are:—pain over the stomach, gall-bladder and umbilicus increased by appendiceal rather than by local pressure; hyperæsthesia over right edge of rectus, dull or colicky pain on exercise* and *pain* associated *with visible peristalsis in the ileo-cæcal region* (obstructive adhesions). Right-sided *dysmenorrhœa* following an acute illness suggesting appendicitis, *diminished power of endurance* and *persistent malnutrition* are frequently present and a *dull aching* or even *colicky, appendiceal pain* may come on *at the height of digestion.* It should be borne in mind that chronic *appendicitis is frequently associated with movable kidney, chronic indigestion, obstinate constipation, alternating diarrhœa and constipation and mucous* (membranous) *colitis.* Further, that symptoms of *obstruction* may be caused by an old appendicitis with dense adhesions. A somewhat extended observation has convinced the author that no surgeon or internalist can hope to recognize all of these cases without an exploratory operation.

Obscure.

Often misnamed.

Fever.

Symptoms suggestive.

Misleading associations.

Differential Diagnosis of Chronic Appendicitis.—This must depend upon the factors already given. In most instances the past history and the acute or subacute attacks are the determining factors.

ACUTE INTESTINAL OBSTRUCTION (see pain—abdominal, p. 64).

ACUTE PERITONITIS.—This may be *local, diffuse or general* and its causes are too numerous for detailed description, but resolve the cases into *primary* and *secondary* groups. The so-called primary or idiopathic peritonitis is a dubious clinical entity. *Secondary* peritonitis means an extension of inflammation from any structure to the adjacent peritoneum. Thus it may arise from traumatism including operation, from extension, in the severer inflammation of the intestinal tract,

Primary rare.

*In children primary operation is usually indicated if the diagnosis can be made within the first 24 hours.

Etiology. from perforation due to absc ss or ulcer, and from chronic ailments such as cancer and tuberculosis. Furthermore, the lowering of vitality in certain chronic diseases seems to render the peritoneum less resistant to the action of germs. The evidences of infection of the peritoneum

Misleading forms. are extremely varied, certain cases associated with profound collapse and death subsequent to abdominal operation showing no lesion save perhaps points of congestion. In some there is little fluid or fibrinous exudate, in others a large amount of fluid and exuded lymph, the fluid being sero-fibrinous, purulent, putrid, or, in wound infection carcinoma and some tuberculous cases, hemorrhagic. **Symptoms.**—In general

Usual type. peritonitis of the perforative or septic type, the *facies assumes the Hippocratic type*, there is usually *severe initial pain* and evidence of *marked prostration or collapse, fever may be preceded by chills* and rise rapidly to a high point or *chill* and *fever* and *pain* be absent. *Continuous high fever is seldom seen.* Painful vomiting and frequent mic-

Urine. turition are common. The *urine is scant, indicanuria is marked*, the

Attitude. *pulse is small and tense*, and the *position is rigidly dorsal*, with the knees drawn up and the *abdomen tense, usually distended* but occasionally

Vomitus. flat, the *vomitus is yellow and bile stained* or later pure green or even

Wasting and tenderness. brownish black and of *fecal odor*. Wasting is rapid, distension tends to increase, tympany being marked, and there is *general tenderness* of an extreme degree. It should be emphatically stated that obliteration of splenic and hepatic dulness is not confined to *perforative* peritonitis but may occur in any extreme case of tympanites. Dulness in the

Fluid. flanks is usual, indicating fluid, and the difficulties of diagnosis in these cases are chiefly related to the identification of the primary lesion. "*Hysterical peritonitis*" may however produce a typical clinical picture

Various confusing conditions. on inspection, but fever, wiry pulse and indican increase are usually absent. Obstruction of the bowel offers great difficulty at times and demands a careful consideration of the factors stated on page 64. Ruptured tubal pregnancy, acute hemorrhagic pancreatitis and various other lesions may demand the surgeon's knife for differentiation.

LOCALIZED PERITONITIS.—In the male the most frequent

Appendiceal or pelvic. cause of localized peritonitis is appendicitis, in the female tubal or ovarian inflammation. A great number of localized inflammatory conditions of the viscera give rise to limited areas of peritonitis, the degree of inflammatory change and its nature depend-

Other causes. ing upon the primary cause. An extended discussion of these forms is out of the question. *The lesser peritoneum*, however, must receive special attention because of its frequent involve-

ment, its importance and the obscurity of its symptoms. The lesser cavity is bounded in front by the gastro-hepatic omentum, the anterior layer of the great omentum and the stomach; below, by the upper layer of the transverse meso-colon, and extends from the splenic to the hepatic flexure of the colon and from the hilus of the spleen to the foramen of Winslow. Above lies the transverse hepatic fissure and that portion of the diaphragm covered by the lower layer of the right hepatic lateral ligament. In the presence of inflammation its communicating opening leading to the greater cavity (foramen of Winslow) may be shut off so that inflammatory exudate may cause a tumor in the upper abdominal quadrant. This is markedly affected by inflation of the stomach which tends to obscure or obliterate the dull note or even the palpation outline of the tumor, and an inflated colon does not cross it, but lies below it. This is properly known as **"subphrenic abscess,"** its most frequent cause being perforating gastric ulcer. Duodenal ulcer and appendiceal abscess account for nearly all of the remainder. **Differential Diagnosis.**—The *symptoms are usually abrupt and associated with pain, vomiting, dyspnœa and signs of sepsis.* The *tumor may be palpable* in some portions of the upper quadrants, *cyst-like in feel, markedly obscured by stomach inflation, and not crossed by an inflated colon,* and when the accumulation is immediately beneath the diaphragm the *symptoms strikingly resemble empyema and the abscess may rupture through the lung, the pus appearing in quantity in the sputum.* (See also hepatic abscess.) The author believes that such cases may in many instances be differentiated by the absence of respiratory lateral displacement of the heart (see page 134). *If air be present, pneumothorax is simulated, but should be easily distinguished upon X-Ray examination by the high position of the diaphragm and underlying shadow.* The mortality in this condition is enormous (80%+).

Boundaries of lesser cavity.

Effect of adhesion.

Stomach inflation.

Offers great difficulties.

CHRONIC PERITONITIS.—The plastic lymph associated with all peritoneal inflammations, acute or chronic, tends to form adhesion bands of greater or less extent, density and permanence, and movable viscera often drag agglutinated surfaces apart to form false bands. It should also be remembered that in *any condition simulating local peritonitis the abdominal wall lacks the persistent general or localized rigidity of the true ailment.* In both acute and chronic forms moreover the ease with which the inflammation may spread from peritoneum to pleura and vice versa must be taken into account. As Edmund Owen says, "the peritoneum acts in inflammation much like an inflamed joint," i.e., it seeks functional rest and is exquisitely tender, *whence*

Adhesions.

Valuable point.

Spreading inflammation.

Tenderness.

Friction.

Misleading signs.

arises the characteristic rigidity, constipation and localized tenderness and the fact that pressure which relieves ordinary colic is intolerable in peritonitis. Further, as in pleurisy, *auscultation may reveal areas of friction* particularly in the region of the subdiaphragmatic organs which move in inspiration (chiefly the liver and spleen). *Chronic adhesions* in the form of false bands may cause intestinal obstruction but in the ordinary form they may be symptomless or give rise to misleading and troublesome symptoms, as in two cases recently observed by the author in which a chronic appendicitis with acute exacerbation was exactly simulated by an extensive adhesion in its neighborhood, the appendix being normal.

PROLIFERATIVE PERITONITIS.—This is characterized by marked thickening without extensive adhesions and *is usually associated with hepatic cirrhosis, more rarely with chronic passive congestion, tumors and similar conditions.* The effusion is usually small, the viscera may be included in the thickened layers and the peritoneum may

Transverse tumor.

be divided into several chambers. Shrinkage of organs may be produced and the rolled omentum usually forms a transverse *tumor at the lower border of the stomach. Nodular growths simulating tubercles or malignant nodules may be present.*

TUBERCULOUS PERITONITIS.—This condition varies exactly as do pulmonary lesions, *there being an acute miliary, chronic ulcerative*

Seek a primary focus.

and *chronic fibroid form.* It may be primary or secondary being frequently associated with intestinal tuberculosis or disease of the Fallopian tubes (30–50% of adults). *The prostate or seminal vesicles may be primarily involved.* The incidence as regards *sex* is as two to one in the male as compared with the female. As regards age it is most frequent in the 3rd, 4th and 5th decade. As regards *race,* more common in the colored than in the white. Traumatism, cirrhosis of the liver and

Obscure cases.

hernia are associated conditions in many cases. **Symptoms.**—As with tuberculosis elsewhere the disease may be *symptomless* and *is usually less pronounced than other forms of peritonitis;* more rarely the onset and course are extraordinarily severe. *In general, there is tenderness, localized or diffuse, and a temperature sometimes continuous, more often*

Usual type.

hectic in type, usually of slight range, and, a *moderate, often hemorrhagic*

Pigmentation.

ascites. Deep pigmentation of the skin is sometimes observed and *tumors are encountered due to inclusion and contraction of intestinal coils, encysted exudate and adherent omentum,* or, *in children, to enlarged mesenteric glands.*

Differential Diagnosis.—Cases with acute onset and violent symp-

toms may be unrecognizable as tuberculous; those with continued fever are differentiated from typhoid by the absence of the Widal reaction and the continued development and predominance of peritoneal symptoms. *Omental tumors are usually transverse and somewhat characteristic in the shape and feel. The presence of an exudate greatly assists diagnosis.* In most instances a suggestive family and personal history or the existence of tuberculous pulmonary, intestinal or tubal symptoms assist differentiation.

Varied types.

Tumor and exudate.

CANCER OF THE PERITONEUM.—Secondary cases need no special description, they take the form of growths, generally distributed and varying from the miliary nodule to large umbilicated growths. The omentum is frequently involved and forms a peculiar tumor as in tuberculosis. Primary disease is extremely rare and most often endothelioma.

Diagnosis.—A positive diagnosis is easy in secondary cases if the primary source be manifest, but is always difficult in the rarer primary forms, indeed a positive differentiation is hardly possible without exploration, the symptoms being almost identical with those of tuberculosis or echinococcus infection in most instances.

Often impossible.

DISEASES OF THE PANCREAS.

ACUTE HEMORRHAGIC PANCREATITIS.—Etiology.—Infection through the gall-bladder and ducts is the chief source of this disease, though bile itself, if forced back into the pancreas by obstruction to its normal outflow may produce fat necroses and hemorrhage. It may also be involved in septicæmia, by traumatism, or secondarily, in the extension of inflammatory processes from neighboring organs.

Symptoms.—*These closely simulate perforation particularly that of duodenal or gastric ulcer, the onset being sudden with violent colicky pain in the epigastrium, associated* with *vomiting* and *collapse*, with *subsequent epigastric swelling.* It is evident that a positive diagnosis is often impossible. The maximum of tenderness *may* clearly follow the outline of the pancreas and there may be a suggestive history of antecedent disease of the gall-bladder. **Diagnosis.**—The diagnosis must be based upon the *sudden onset* with pain and vomiting, symptoms of *collapse* and the appearance of a fixed *tumor* corresponding to the position of the affected organ. The diagnosis is usually though not always made by exploratory section. The urine may show leucin and

Violent onset.

Surgical diagnosis usually.

tyrosin after treatment with basic lead acetate, filtration, treatment of the filtrate with hydrogen sulphide and evaporation. **Prognosis.**—Recovery is the exception.

ACUTE SUPPURATIVE PANCREATITIS (pancreatic abscess).—Suppurative pancreatitis is associated with preliminary digestive disturbance, fever, possibly of a septic type and what is more important by a fixed epigastric tumor.

GANGRENOUS PANCREATITIS may result from hemorrhage or rarely suppurative inflammation. It has no distinguishing symptom aside from those of the primary hemorrhagic or septic pancreatitis, followed by collapse and death in a few weeks or days. The distinction is made post mortem.

CHRONIC PANCREATITIS closely resembles *histologically* in its two forms, atrophic and hypertrophic cirrhosis of the liver, the former condition is frequently associated with diabetes.

PANCREATIC CYSTS.—The chief characteristics are recurrent colic associated with nausea, vomiting and progressive abdominal distension, glycosuria, emaciation and a fixed median tumor, cystic in feel, and *lying above the inflated colon and below the stomach.* They may attain great size.

CARCINOMA OF THE PANCREAS.—Primary carcinoma involves usually the head of the gland. Metastasis or direct invasion of other organs is frequent; the condition is rare. **Symptoms.**—These are chiefly those of *progressive emaciation, anæmia, intractable dyspepsia,* and *dull or severe epigastric pain,* the *stools are often light and pasty, muscle fibres of ingested meat may result* from faulty pancreatic secretion and jaundice and ascites are not infrequent. The tumor is median unless the tail of the pancreas be the part involved and glycosuria, jaundice and various pressure symptoms may be present

PANCREATIC CALCULI.—The rough round or spiculate stones are, in rare instances, associated with violent epigastric colic and may be found in the stools. Ordinarily they produce an interstitial pancreatitis or lead to abscess.

DISEASES OF THE LIVER AND THE BILIARY PASSAGES.

CONGENITAL ANOMALIES.—An extraordinary variety may be found including abnormalities in the size, number and position of the lobes, the presence of accessory masses of tissue, complete absence of the organ, abnormal mobility, and even its displacement to the left

side, as is seen in complete *situs inversus*. It has been found within the thorax in cases of diaphragmatic hernia, or following injury, and may be markedly displaced downward, or grooved, swollen, and thickened by tight lacing. Multiple depressions of a similar sort may appear upon its superior surface, and are not satisfactorily explained. The gall-bladder may be entirely lacking and curious variations occur in the size, number and distribution of its ducts.

Corset liver.

INFLAMMATION OF THE LIVER (*acute hepatitis*.—**Etiology.**—Inflammation may result directly from injury or follow an infection, diseases of the stomach and intestines or portal veins, and may be either simple or suppurative in character.

PYEMIC ABSCESS OF THE LIVER.—It will be readily understood that pyemic abscess, whether single or, as is usual, multiple, may result from infection conveyed, directly or indirectly, through arterial, venous, or lymphatic channels. Any form of pyemia or general septic absorption may result in infective embolism of the hepatic artery, a great many of the suppurative and ulcerative lesions of the abdominal tract result in portal embolism and almost any of the acute infectious diseases in their severer forms may produce hepatic abscess. Amongst the vast number of associated conditions may be mentioned ulcer and cancer of the stomach and duodenum, inflammation and malignant growths involving the pancreas, gall-bladder or its ducts, and, any ulcerative and suppurative process in and about the intestines, such as typhoid fever, dysentery, appendicitis, cystitis, prostatitis, tuberculosis, etc. Caries of the spine and abscess in and about the kidneys may also cause hepatic abscess, as may the echinococcus, the Balantidium coli, ascarides and liver flukes. The so-called tropical abscess is almost invariably associated with amœbic dysentery and such an abscess is ordinarily solitary (75%).

Usually secondary.

Associated conditions.

Tropical abscess.

Symptoms.—These, usually pronounced and localizing, more rarely latent and obscure, are chiefly:—enlargement of the hepatic area or localized swelling, pain, and the well known symptoms of *sepsis*. The pain is not usually severe until the abscess approaches the surface and is not distinctly localized, but felt both over the liver and in the region of the right shoulder blade and in some instances, chiefly at a point to the left of the median line, below the costal margin. *Enlargement of the liver is an invariable symptom and is usually marked;* hence both the upper and lower hepatic borders, should be examined and the condition of the lower interspaces investigated. *A localized swelling* corresponds to the "pointing" of the abscess, is invariably accompanied

Usually pronounced.

Pain variable.

Localizing signs.

Edema.

by edema of the superficial tissues, and in some instances may show fluctuation. Auscultation may reveal *peritoneal friction*, due to capsular involvement and precisely similar to pleuritic friction, the tendency being to form adhesions and limit movement. As might be inferred *loss of strength and weight* is marked, and *loss of appetite, morning vomiting*, and in general, *pronounced dyspeptic phenomena. Fever* is either definitely intermittent or decidedly irregular, and is usually associated with chills, and severe nocturnal sweats. *Jaundice* may or may not be present and no stress should be laid upon its absence. It is said that the decubitus of such a patient is characteristic, the right knee being drawn up, the right shoulder lowered, but this might apply to any disease affecting this particular region. **Complications.**—The only complication needing attention is *perforation.* A knowledge of the anatomical structure enables one to understand *in how many directions and with what serious results, an hepatic abscess may burrow and discharge its contents.* Such perforation may occur outward through the skin, after the formation of adhesions, either directly or, more frequently, through a sinuous burrow. It may perforate into the pleura and cause empyema, and indeed may cause purulent pleurisy with direct perforation and abscess of the left lobe, or, perforate the pericardium, while perforation into the peritoneal cavity is very rare, as is that involving the portal vein, the inferior vena cava and the right kidney. Most practitioners have met with cases of direct perforation with intervening empyema through the lungs and bronchi. The discharge of pus by the bronchus is usually preceded by severe cough, and the expectoration of bright red sputum and local examination will indicate merely a limited area of pneumonic infiltration. Sometimes it may be that the findings are quite negative, yet, shortly after, the patient will in a violent fit of coughing, raise a considerable quantity, oftentimes a large amount, of pus, intermixed with blood, often brown sero-purulent or, bile stained, not infrequently containing shreds of lung tissue. Strange to say these cases may go on for long periods with a daily expulsion of quantities of pus, in one case reported over a pint, and yet spontaneously subside, leaving only a certain amount of lower chest deformity and crippled lung base; the aid of modern surgery is however always needed.

Adhesions.

Sepsis.

A variable sign.

Sequelæ of perforation.

Pulmonary form.

Differential Diagnosis.—Too often these cases are misconstrued or entirely overlooked. Stress should be laid upon the *existence of causative factors.* Furthermore, *enlargement of the hepatic area is invariable. Jaundice* when present is extremely suggestive, and *local-*

Error frequent.

ized swelling, whether over the free surface of the liver or the lower right interspace is most important. *A distinctly septic trend of the symptoms at once suggests abscess. The spleen is enlarged* and there is marked *leucocytosis* which may exceed 50,000. In empyema, there is usually an antecedent history suggesting that condition and the author's sign of unilateral pleuritic exudate (see page 134) should prove helpful, as should the character of the pus withdrawn by aspiration. As between hepatic and subdiaphragmatic abscesses, a surgical exploration is often required for positive differentiation.

Blood.

Fluoroscope.

Exploration.

Prognosis.—The prognosis depends upon three factors, i.e. the number of abscesses, and, the promptness of diagnosis and surgical relief. Cases of multiple abscesses are practically hopeless.

HEPATIC HYPERÆMIA.—Transient active hyperæmias are unimportant and we have to do essentially with *chronic hyperæmia*, which is invariably due to some cause tending to prevent the free flow of blood from the liver into the vena cava. Amongst the latter causes may be mentioned chronic heart disease, especially such as results in dilatation of the right heart, asthma, emphysema, bronchiectasis, pulmonary fibrosis and portal thrombosis. Such a congestion, typically shown in cases where the tricuspid valve has given way, results in the production of the so-called nutmeg liver, here being central lobular congestion with peripheral anæmia associated with fatty degeneration. The liver is large, though in the presence of long continued congestion it may undergo a certain amount of contraction attended by the formation of a finely nodular surface, and section shows dark red markings on a light background, from whence arises the name "nutmeg liver."

Secondary.

Nutmeg liver.

Symptoms.—The essential symptom is *increase in size* as recognized by palpation or percussion, with especial reference to the lower border, or lacking this, increased density associated with a certain amount of pressure tenderness particularly over the left lobe, the secondary symptoms (such as chronic gastric catarrh and intestinal disturbance), and the existence of a primary cause. Ascites may or may not be present, according to the degree of change produced and hæmatemesis is rare.

TUMORS OF THE LIVER.—Primary malignant disease of the liver whether sarcomatous or carcinomatous is excessively rare. Secondary carcinomatous growths are common, sarcoma most unusual.

Primary rare.

Common.

Secondary Carcinoma.—Most often follows involvement of the stomach, pancreas, kidney and intestines, and usually first appears in the right lobe. As the process advances, the nodular masses tend to soften and become umbilicated by central absorption, this condition

Nodules.

being oftentimes distinctly palpable. **Cystic Tumors** are rare, and usually small. They ordinarily originate in the gall ducts, and yield a clear or slightly yellow fluid.

PARASITIC INVOLVEMENT.—The amœba coli is the cause of practically all tropical abscesses, being found sometimes, even in the absence of dysenteric symptoms. The echinococcus is the most important of the various other forms of parasites encountered.

ECHINOCOCCUS CYSTS.—These are due to the embryo of the tænia echinococcus, a tape worm, which is rarely encountered save in Ireland and Australia, though found occasionally in Germany, and now and then met with amongst the Icelanders who have settled in British Columbia. The worm is not more than a quarter of an inch in length, and belongs properly to the dog. It is composed of three or four segments, the mature terminal portion being about 2 mm. long and 0.6 mm. wide, yet it contains several thousand eggs. The head shows a rostellum, 4 suckers and 2 rows of hooklets; the embryo provided with six hooks and arranged in pairs, makes its way through the intestinal wall and may enter the portal vessel and be carried from the liver and the systemic vessels by which it reaches the brain or other distant organs, or it may remain in the peritoneal cavity or the surrounding muscles. It is thought that this embryo may find entrance through infected dust as well as through drinking water and in Australia and Iceland where the disease is most prevalent, the dogs are very generally infected and the relation between them and their masters is very close.

Habitat.

The parasite.

Points of entrance.

Conveyance.

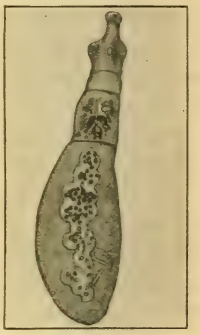


Fig. 121.—Tænia Echinococcus. (Braun.)

General Symptoms of Hydatids.—Diagnosis primarily depends upon the presence of a cystic tumor and upon the recovery of hooklets from fluids discharged or obtained by puncture. The presence of hydatid disease in other portions of the body is of course suggestive. In the liver, the symptoms are more distinct, owing to the fact that these cysts when superficial give upon percussion a peculiar vibration known as the "hydatid" thrill. See page 230. They may be large, and, in this location, produce various pressure symptoms, or pyemic symptoms should they suppurate. Such cysts like hepatic abscesses may rupture in almost any direction.

Tumor.

Hooklets.

Percussion thrill.

CIRRHOSIS OF THE LIVER.—The central factor in all forms of cirrhosis of the liver is the increase of connective tissue, and the term is a general one, applied to similar changes in other organs. We have clinically to deal with four varieties. (1st). **Laennec's cirrhosis;** the common form of the disease as encountered in every clinic. (2nd). **Hanot's cirrhosis.** (3rd). **Syphilitic cirrhosis.** (4th). **Chronic perihepatitis** or capsular cirrhosis. The cirrhosis due to chronic disease of the heart or lungs, (nutmeg liver), is discussed under chronic hyperæmia, and large numbers of minor forms of some pathologic and etiologic interest need not be discussed in this volume. **Etiology.**—Certain of the infections, both acute and chronic may result in cirrhotic changes. Chronic obstruction of the bile ducts, chronic passive hyperæmia and anthracosis are to be numbered amongst the etiologic factors. Alcohol is the one really important cause of Laennec's cirrhosis, the form of predominant interest and frequency of occurrence, and information concerning the extent and duration of alcoholic indulgence is prerequisite to diagnosis. Gout, lead poisoning and the excessive consumption of highly spiced and over-rich articles of food, though contributive factors, are seldom of primary importance. It follows then that lacking a history of prolonged use of alcohol, a positive diagnosis of Laennec's cirrhosis will often prove erroneous, while on the other hand, a knowledge of previous alcoholic indulgence, even though moderate, may suggest the proper explanation for many obscure gastro-intestinal symptoms. **Morbid Anatomy.**—*True atrophic cirrhosis*, or "gin drinkers' liver" is small, hard, resistant to the knife, and irregularly lobulated or nodular. The little nodules may, by the contraction of the fibrous bands, become prominent, and thus form the "hobnail liver," so called from its resemblance to the hobnail shoe. The color is yellow, and the cut surface is mottled with color running from yellow to brown. Microscopically, the changes consist in the formation of new connective tissue around the sheath of the veins, and this, contracting, causes compression of the portal capillaries, portal obstruction, and, therefore, congestion of all the territory normally drained by the portal vein. *It is this chronic passive congestion which produces most of the clinical symptoms of the disease*, such as ascites, chronic catarrhal gastritis, enteritis, hæmatemesis, bleeding hemorrhoids, and, that distension of the superficial abdominal veins known as the Caput Medusæ, this last symptom appearing, usually, if at all, after the oncoming of ascites, which is a direct and important contributory factor. **Hypertrophic Cirrhosis.**—Called also hepatog-

Four varieties.

"Nutmeg liver."

Many factors.

Alcohol the chief.

A diagnostic aid.

"Hobnail liver."

Source of symptoms.

Biliary form rare.

enous and biliary, it is a comparatively rare form, and is characterized by a general increase in size and less firm resistance to section than is present in the atrophic form. Both the outer and the cut surfaces are comparatively smooth and the term unilobular as opposed to multilobular is applied to this condition, because of the involvement of individual acini rather than of groups. The process is practically a pericholangitis and there is much evidence to support the theory of direct infection as the primary cause. **Perihepatitis or Capsular Cirrhosis.**—This is merely a chronic capsulitis, the liver substance taking little or no part in the process. The same process in the spleen and peritoneum is said to be an almost invariable accompaniment, as is chronic interstitial nephritis. Osler describes two classes of cases, one associated with the ordinary symptoms of cirrhosis, the other with adherent pericardium and chronic mediastinitis.

Probably an infection.

Symptoms of Laennec's Cirrhosis.—A diagnosis of the ordinary form of cirrhosis of the liver depends ordinarily to an unusual degree upon the knowledge of the patient's habits and the proper interpretation of secondary symptoms. *Given any case presenting a history of immoderate or even moderate alcoholic indulgence extending over a period of many years and showing symptoms of chronic gastric catarrh, chronic catarrhal enteritis, even without hemorrhoids and hæmatemesis, the diagnosis of an existing hepatic cirrhosis should always present itself. The disease is seldom recognized until well advanced, but this is no doubt due in many instances to a failure to appreciate the value of grouped diagnostic factors.* The physical signs of the disease may or may not be well defined. By auscultatory and direct percussion and by palpation one may usually make out a decided shrinking in the hepatic outline, and, especial stress should be laid upon the fact that the *early shrinkage is most marked over the left lobe. The spleen is moderately enlarged* in 75 or 80% of all cases, another factor of great importance. The so-called pre-atrophic stage of engorgement is so seldom positively determined that it might as well be discarded as unproven. Another fallacy is that pertaining to the direct recognition by palpation of the hobnail projections on the liver surface. These can rarely be felt, and no stress should be laid upon negative findings. *When the liver can be palpated, the sharp firm edge indicating induration is most suggestive and valuable.* A co-existent ascites ordinarily makes it impossible to accurately palpate either the liver or spleen and in any such case the best opportunity arises immediately after the aspiration of the ascitic fluid. The so-called "dipping" method

Habits.

Congestive symptoms.

Late recognition.

Physical signs.

Pre-atrophic stage.

Value of palpation variable.

so often recommended, which consists in burying the fingers by a sharp stroke upon the abdominal wall in order to displace the fluid and meet the descending liver edge in inspiration is usually futile. "Dipping."

The Caput Medusæ, so called from its fanciful resemblance to the Medusa head is formed by the enlargement of the superficial lateral veins of the abdomen, and when present is a symptom of much interest and importance. Every clinician must realize, however, that *it is a comparative rarity*, moreover, it is often confounded with the general distension of the superficial abdominal veins met with in various conditions producing pressure and obstruction in the abdominal circuit. It should be kept by itself as a clearly defined symptom, as the veins which represent the Medusa locks are the lateral veins joining the axillary and the femoral, and possess a curious valvular arrangement, which normally directs blood flow from above the umbilicus to the axilla, and, below it, to the femoral veins, but, in the presence of any marked obstruction, permit a direct upward flow. To summarise:—the diagnosis of atrophic cirrhosis must rest upon some or all the following data:—(a). *A history of high living, over-feeding, and in nearly every instance alcoholic indulgence.* (b). *Loss of appetite, morning nausea and vomiting, irritable stomach, excessive fermentation, constipation or intermittent diarrhœa, and various other symptoms of chronic gastric or intestinal catarrh.* (c). *The recognition of a shrunken, hardened liver and enlarged spleen.* (d). *Loss of weight, strength, activity and color* (a ruddy color *may* persist until late in the disease). (e). *Hemorrhoids*, blind or bleeding. (f). *Ascites.* (g). *Hæmatemesis.* (h). *Jaundice.* This last is an unusual symptom in atrophic cirrhosis, though sometimes met with during the last months in a form so extreme as to produce a color strikingly like that seen in Addison's disease. It must never be forgotten that *the ascites of atrophic cirrhosis ordinarily precedes any marked general edema or anasarca* and remains usually for a long period quite alone or associated with a moderate edema of the lower extremities due to pressure exerted by the ascitic fluid. These cases furnish some typical examples of the "poached egg belly."

Seldom marked.

Veins involved.

Diagnostic summary.

Edema slight.

Prognosis.—The disease is slowly progressive and beyond therapeutics though the cessation of the use of alcohol, or of any other habits of a similar nature, and a thoroughly hygienic life will do much to lengthen the patient's term.

Incurable.

Differential Diagnosis.—*Simple atrophy of the liver.* This condition accompanies various cachexias, characterized by profound exhaustion and marasmus, is suggested by such conditions, and, is of no clinical

Unimportant.

interest or importance. *Nutmeg liver.* An atrophic nutmeg liver may give rise to much difficulty, but the preexistence of disease of the lung or heart, and the fact that it does not present a primary ascites, but one secondary to a general anasarca, usually suffices for its differentiation. *Fatty cirrhotic liver* A very considerable number of the cases of cirrhosis that we meet with are fatty livers, a form easily recognized by palpation, the organ being enlarged and of firm consistence. It presents no essential difference in symptomatology from that of the ordinary atrophic form. *Portal phlebitis and thrombosis.* The diagnosis of this condition depends chiefly upon the existense of a potent cause such as ulceration, gall stone and suppurative diseases and generally the condition is suggested by a *very rapid onset of ascites*, splenic enlargement, and the other symptoms of portal obstruction, together with their *rapid* recurrence after paracentesis, though this sometimes occurs in true cirrhosis. The specific gravity of the *clear* ascitic fluid is nearly always below 1015 in the case of cirrhosis, whereas, in chronic peritonitis it is distinctly turbid, and in ovarian cyst, of higher specific gravity and containing a greater amount of albumin. **Hyperthropic Cirrhosis** (Hanot's disease, biliary cirrhosis).—This is a comparatively rare ailment, and can hardly be confounded with true cirrhosis, the spleen is enlarged, icterus is intense, and the secondary symptoms of portal obstruction are entirely absent, ascites if occurring at all, coming on late as a part of the general cachexia. The color of the stool shows an intermittent bile secretion, the urine is abundant as contrasted with that of atrophic cirrhosis, and contains bilirubin. The disease is one of long duration, lasting on the average from five to ten years. **Syphilitic Cirrhosis.**—This disease may assume either an atrophic or hypertrophic type and represents a genuine interstitial syphilitic hepatitis. As seen in the adult it is atrophic, in children, ordinarily hypertrophic. A positive diagnosis is never possible, but is rendered probable by the history or marked evidence of syphilis. **Syphilitic Gummata of the Liver.**—These may occur at any age, as a result either of congenital or acquired syphilis, and, varying greatly in size. The clinical picture is that of hepatic cirrhosis, but after the removal of any ascitic fluid that may be present, the nodular outline of the liver is often appreciable. Certain cases show a peculiar latency as regards general symptoms, though representing marked and easily recognizable tumor. *The most important clinical inference is that when one meets with a nodular liver, with or without symptoms of cirrhosis or history of syphilitic infection, he should apply the therapeutic test of*

Readily recognized.

Often seen.

Suggestive course.

Exudates and transudates.

Rare.

Marked icterus.

Urine.

Two forms.

Nodules.

Latent cases.

Therapeutic test.

mixed treatment, and indeed this should be applied in practically all cases of solid growths in the abdominal region. **Amyloid Liver** (amyloid degeneration of the liver).—This condition is invariably secondary to some severe derangement of metabolism, especially those associated with chronic septic absorption, as caries of bones and pulmonary tuberculosis of the advanced type. It may also be the result of chronic and neglected syphilitic infection. The liver is large, smooth, and all symptoms of portal obstruction are absent. Icterus is seldom present. The disease is invariably fatal.

Secondary.

Large smooth liver.

CATARRHAL JAUNDICE (*acute catarrh of the bile ducts*).—This is essentially a part of a catarrhal gastro-duodenitis and is actually an obstructive jaundice, due to inflammatory products and swelling. It is especially common in children but may be met with at all ages in connection with acute or subacute indigestion or a large number of diseases associated with a tendency to catarrhal inflammation of the upper intestinal tract. It sometimes seems to be epidemic and due to acute infection. Save for the evidence of jaundice (color, light pasty stools, etc.) it may be wholly symptomless, or, especially in the epidemic form, there are symptoms of indigestion associated with mild fever and general malaise. The liver is usually slightly enlarged but the spleen rarely tumefies. *Duration* is from 2 to 12 weeks. *Chronic catarrhal jaundice* is merely an expression of chronic obstruction, due to any of the causes described on the preceding page. It may be complete or incomplete, persistent or intermittent, and will be discussed further under cholecystitis and cholelithiasis.

Icterus.

Epidemic form.

Mild symptoms.

ACUTE CHOLECYSTITIS.—In from 15 to 20% of the cases a cholecystitis exists without gall stones or other causal indications. The flora of the disease include a pyogenic staphylococcus, steptococcus, pneumococcus, colon and typhoid bacillus.

Symptoms.—It should be stated emphatically that *jaundice may or may not be present and that the pain may be distinctly localized in the region of the tip of the 9th costal cartilage, over the gall-bladder itself, or, may first be referred to the epigastrium or even to the region of the appendix.* Pain is also felt usually under the right scapula or even in the right shoulder. There are nausea, vomiting, rapid pulse, fever, prostration, local tenderness and rigidity which in most cases become definitely localized.

Jaundice and pain, variable.

CHOLELITHIASIS.—Gall stones may be single or present by the hundred or thousand (usually from 5–10 or 12 are met with). They may

Number.

Size and form.

vary in size from mere grains to stones measuring six inches or more in length, are polygonal with smooth facets, more rarely smooth, ovoid or round. They have a nucleus, usually of bile pigment and germs have been reported and undoubtedly are actively concerned in the processes leading to their formation. Section shows laminated structures with radiations and an analysis reveals biliary and fatty acids, magnesium and calcium salts and even traces of iron and copper. Cholesterin constitutes the bulk of the stone, their color is brown and the surface may be rough and mulberry like. Autopsies show an astonishingly large number of cases of gall stones* and have proven that during life they have in many instances yielded no symptoms save probably "*indigestion*," "flatulence" and the like. Ordinarily their migration into the ducts gives rise to biliary colic. This is characterized by *violent and abrupt pain in the right hypochondriac region or epigastrium, radiating to the shoulder blade with vomiting, sweating, circulatory depression and usually intermittent fever which may be preceded by a sharp chill.* The localized *tenderness* and muscular *rigidity* tend after a time to *become localized in the region of the tip of the ninth costal cartilage*, the liver and spleen may be enlarged, and the urine frequently contains albumin. If the stone be in the cystic duct there is no jaundice, or but a suggestion of it indicating complicating cholangitis; in the common duct, persistent and severe obstructive, or, slight transient jaundice.† These attacks may be repeated until the stone passes onward or backward or is removed by operation, and death rarely occurring during an attack, though phlegmon, rupture and general peritonitis are possible. The location of the pain in the upper abdominal and lower thoracic segment with localized tenderness (best elicited by deep pressure in full inspiration) and perhaps a history of previous attacks are the chief distinguishing symptoms. If jaundice co-exists the diagnosis is almost absolute, yet the author has reported a case of floating kidney, associated with Dietl's crises during which jaundice occurred and operation revealed a normal gall-bladder. A second case of floating kidney in which the same condition without jaundice existed and proved to be due to gall stones. A case more recently observed showed no tenderness over the liver or gall-bladder

Structure and constituents.

Latent cases.

Colic.

Tenderness.

Cystic duct vs. Common duct.

Chief symptoms.

Dietl's crises.

Illustrative case.

* Autopsies also demonstrate the tendency of unoperated cholelithiasis to lead to carcinoma of the gall-bladder—the latter disease being seldom found without the former—but this does not apply to operated cases.

† Many stones are of the "ball valve" variety, persistent obstruction not being maintained.

even under forcible deep upward pressure in deep inspiration and the pain was invariably *left sided.* The stools should be carefully examined during and immediately after a supposed attack. Gall stones, cholecystitis or adhesions may alike give rise to recurrent attacks of more or less severe pain and an old gastro-duodenal ulcer may through the same cause be associated with symptoms hardly distinguishable from those of gall stones.

Stools.

Misleading conditions.

Obstruction of the Common Duct.—Obstruction may be absolute with profound and persistent jaundice (pale stools, etc.) and be due to one or several stones, or it may be only partial with remittent jaundice or a so-called ball valve stone producing paroxysmal chills with fever and sweating, persistent but variable jaundice, intensified during the paroxysm, hepatic pain and indigestion. Suppuration may or may not complicate common duct stones and empyema of the gall-bladder and even hepatic abscess or perforation may occur. It would be indicated by a more persistent fever, remittent rather than intermittent, more marked enlargement of the liver and leucocytosis.

Septic crises.

Ball valve stones.

Septic angiocholitis.

Cystic Duct.—The chief features of cystic obstruction are the *absent or slight jaundice,* the distension of the gall-bladder, which may reach an enormous size, and, the common occurrence of a complicating cholecystitis occasionally of the suppurative type with empyema of the gall-bladder. Calcification of the walls and atrophy *may* occur though less frequently than in common duct stones; in some cases is so complete as to leave hardly a trace upon superficial examination, as in a case seen by the author in which a careful dissection was required and revealed the presence of a large number of stones apparently embedded in the hepatic tissue. Fever is less often present than in stone of the common duct.

Enlarged gall-bladder.

Atrophy.

General Comment.—*The utmost difficulty may be experienced in both acute and chronic forms of cholelithiasis or cholecystitis* and the greatest stress must be laid upon the location of the pain and presence of localized tenderness, irrespective of jaundice which of course greatly assists the diagnosis when present. A palpable gall-bladder moves with respiration, has its point of attachment in the region of the ninth costal cartilage and is movable only in the segment of a circle whose centre is its attachment. It should be noted that jaundice of marked degree is absent in obstruction of the cystic duct and a distended gall-bladder is rare in obstruction of the common duct. Stone in the common duct is *usually* associated with a *contracted gall-bladder;* in the cystic duct with distension; in malignant disease blocking the common

Often obscure.

Pain and tenderness.

Tumor.

Jaundice.

Malignant cases.

duct with distension,* profound and persistent jaundice, slight or no pain. *Finally:*—All degrees of *pain, fever and tenderness* may be encountered.

ACUTE YELLOW ATROPHY.—(*Icterus gravis. Malignant jaundice.*)

A clinical curiosity.

Etiology.—This excessively rare disease chiefly affects women and falls mostly upon the third decade. It is frequently associated with pregnancy, has followed profound emotion or shock, syphilis, alcoholic excess and various other ill-determined factors.

Morbid Anatomy.—The liver cells are necrotic, and there is a general catarrh of the finer bile ducts associated with hemorrhages between the liver cells. The organ itself is greenish yellow, greatly shrunken, flabby, with a loose capsule, and on section it is mottled with yellowish brown and red. The heart may show fatty degeneration, the spleen is usually enlarged and the kidneys may also be degenerated.

Progressive icterus.

Symptoms.—The picture is primarily that of a catarrhal jaundice succeeded in a few days or perhaps two or three weeks by vomiting, perhaps bloody subcutaneous and mucous hemorrhage, headache, delirium, muscle tremor and sometimes convulsions. The jaundice deepens with

Variable fever.

the onset of the graver symptoms. There is usually no fever, rarely a

Profound exhaustion.

marked pyrexia. The general condition is that of the typhoid state and the condition terminates ordinarily in coma. Shrinkage of the

Shrucken liver.

liver may greatly reduce or entirely obliterate its normal area of dul-

Urine.

ness. The urine contains bile, frequently albumin, albumose and casts. Leucin and tyrosin are frequently but not constantly present, the stools are clay colored, urea is diminished but ammonia nitrogen is increased.

Differential Diagnosis.—The interesting symptom group differ-

Phosphorus.

entiates this disease from all others except phosphorus poisoning, in

Luminous vomitus.

which the three leading symptoms, viz.:—hemorrhage, profound jaundice and shrinkage of the liver may occur. The examination of the vomitus, and history of the case are the chief distinguishing points. Grave symptoms may occur in connection with other diseases associated with jaundice but the picture of acute atrophy is seldom present.

WEIL'S DISEASE (*acute febrile jaundice*).—This disease is with

Riedel's lobe.

*The linguiform process of Riedel and Cruveilhier (Riedel's lobe) is especially common in chronic disease of the gall-bladder, though occasionally present in other abdominal ailments (Moynihan). It may lie to either side of or over-lay the gall-bladder and is clinically a small freely movable but attached linguiform body—which lacks pendulum motion—may be felt through with the hand at the back and has a sharp edge. (J. U. Goodrich and W. A. Dennis, personal communication.)

difficulty distinguished from an acute catarrhal duodenitis of the severe epidemic type. It occurs chiefly amongst butchers and is ordinarily a disease of warm weather. Affects young and middle-aged adults, and is characterized by early jaundice, tenderness and tumefaction of the liver and spleen, severe headache, and *pain in the legs, arms and lumbar region, and, oddly enough, in the masseter muscles.* The stools are often light as in actual obstructive jaundice. The disease lasts for a week or two, the temperature is septic in type, and may be moderate or high and albuminuria may be present.

Symptoms.

Peculiar feature.

Differential Diagnosis.—The peculiar symptom complex just described, offers the only means of differentiation from the ordinary acute *catarrhal duodenitis. Malaria* in its remittent form would show the parasite in the blood, *phosphorus* poisoning by the history of its ingestion, and the phosphorescent character of the vomit. The disease is rare and seldom fatal.

DISEASES OF THE ESOPHAGUS.

ACUTE ESOPHAGITIS.—Occasionally occurs as a result of the irritation of foreign bodies, the action of corrosive poisons or hot liquids. or, in connection with diphtheria or other acute infections. It may also be secondary to malignant disease or an extension of a catarrhal process in the throat. Its symptoms are chiefly dull substernal pain and dysphagia with or without spasm.

Esophageal Strictures and Diverticula.—In the absence of paralysis, inability to swallow solid or liquid food demands immediate recourse to the esophageal bougie. True stricture is usually due to cicatricial contraction following the ingestion of corrosive poisons, syphilis, or, *a malignant growth* and is rarely congenital. Obstruction from the pressure of *aneurism* or other tumors, or even, it is said, of massive pericardial effusion may occur. *A bougie should never be passed without a careful preliminary examination for possible aneurism or necrotic processes and it should be remembered that* 9 *out of* 10 *strictures in adults are due to malignant growths.* Strictures may be single or multiple, sharply defined or involving the whole tube, but are usually either near the cardia or just below the pharynx. No definite information is obtainable without the use of the bougie, but as a matter of interest the esophageal bruit may be auscultated at the left of the spine. It should be heard at the left of the seventh dorsal vertebra within 7 seconds of the time that a mouthful of water is swallowed, and a delay suggests obstruction. The most convenient bougies are those with detachable

Dysphagia demands exploration.

Beware of aneurism.

Bruit.

Best bougies.

heads of varying size and an elastic steel or whalebone stem. As in the passage of a urethral sound and for the same reasons, the larger sizes should be first tried and *forcible manœuvres avoided.* The patient

Technique.

should be asked to breathe deeply with the head thrown far back and the spine straight, and *without introducing the finger*, the bougie is passed firmly and expeditiously along down the pharyngeal wall disregarding the slight obstruction encountered at the cricoid level. Oftentimes

Fluoroscopy. Perforation.

the exact position of the stricture may be shown by fluoroscopic examination or X-Ray photography in a tolerant patient with the bougie in situ. Perforation during the procedure is a rare but deplorable accident. *Secondary dilatations or diverticula* above the stricture and hypertrophy of the walls most frequently occur on the posterior wall

Site.

near the pharyngeal junction, i.e. opposite the cricoid cartilage and

Symptoms.

there may be no obstructive lesions. Subjective sensations of retention and irregular regurgitation of ingesta lacking gastric juices and often fetid, are the chief symptoms.

Carcinoma of the Esophagus.—Primary carcinoma is rare, most

Usually secondary.

cases of obstruction being due to malignant disease extending upward from the cardia. The symptoms are those of obstruction associated with rapid and extreme emaciation and cachexia. Perforation into

Emaciation.

the mediastinum, aorta, pericardium or pulmonary and pleural structures may occur or the spine may be involved. In some of these cases the X-Ray gives evidence of positive value as shown by the plates on pages 211, 212.

Esophageal Spasm.—This condition is usually associated with functional nervous disease such as hysteria, hypochondria, neura-

Readily detected.

sthenia or more rarely epilepsy and chorea. It is also a symptom of hydrophobia and the diagnosis rests upon the recognition of the causative factors named and the passage of an esophageal bougie of full calibre which meets with only temporary obstruction. Liquids can usually be taken but solid food excites spasm.

Special Instruments.—Many special instruments have been devised for direct inspection, exact calibre determination, measuring the capacity of diverticula, etc., but few have accomplished much in a practical way. A bismuth subnitrate porridge may render diverticula plainly visible by the X-Ray.

URINALYSIS AND DISEASES OF THE KIDNEY.

General Considerations.—*The normal activity of the kidney depends upon a propor performance of function on the part of the other related*

organs, as well as upon the structural and functional integrity of the kidney itself. Its remarkable intrinsic nervous mechanism makes it difficult to apply rigid rules governing its activity, but it may be said that the *quantity of urine and inorganic salts* depend upon osmotic glomerular filtration as affected by blood pressure and volume, whereas the output of *urea and its congeners* is determined by the peculiar selective action of the cells of the convoluted tubules. *Therefore anything affecting the renal structure or circulation, directly or indirectly, must alter the amount and constitution of the urine.*

Filtration.

Selective cell action.

Value of Urinary Examination.—*No other secretion or excretion of the body furnishes so great and so varied information.* It measures variations in nutrition and waste and indicates approximately or positively many diseases, both local and general, poisoning (coal tar products, morphia, lead, mercury, chloral, etc.), Bright's disease, diabetes, spermatorrhœa, cystitis, pyelitis, gonorrhœa, oxaluria, lithæmia, the presence of fever, a failing heart, typhoid fever, pneumonia, septicæmia, urinary and general tuberculosis, filaria sanguinis hominis, dyspepsia, chronic appendicitis, hysteria and impending or established jaundice may be mentioned as some of the conditions. either directly diagnosticated or strongly suggested by urinary signs. The ordinary examination of the urine is largely qualitative, but certain necessary quantitative methods involve only a small amount of apparatus and a negligible loss of time and *all vital procedures may be carried out by simple methods whose margin of error is not so great as to vitiate clinical results.*

Wide scope.

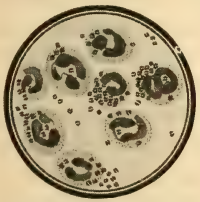


Fig. 122.—Gonococcus.

Qualitative.

Quantitative.

The First Step in the Urinary Examination.—Whenever possible one first obtains the total urine for 24 hours as best representing the functional activity of the kidney as affected by changes in its structure or diseases of related organs. It should be collected in clean bottles, the night urine separate from that of the day. The urine passed on the first morning is disregarded, all voided for the remainder of that day saved and kept in a cool place and that passed after retiring and upon arising the *following* morning separately collected. In certain important renal diseases, notably in interstitial nephritis, the normal ratio of day to night excretion is greatly modified or reversed. Ordinarily, the day urine is three or four times greater in volume than that of the night, hence any marked variation is suggestive. Of individual

Get 24 hours urine.

How collected.

Night vs. Day.

Best single specimen.

specimens, that passed late in the day, preferably after a full meal and exercise, is most likely to contain albumin or sugar. Except in suppurative disease of the kidney or bladder, *that least likely to show abnormalities is the early morning urine.*

Variations.

The normal amount of urine varies from 500–2000 c.c. and this variation may be temporary or permanent, depending upon exercise, the amount of perspiration, the kind and amount of water ingested, or the nervous condition of the individual. The smaller amount represents usually diminished ingestion or increased loss of fluid through other channels such as free perspiration during hot weather, or a diarrhœa. Women drink little water and usually show a small daily total.

Temporary and unimportant.

Polyuria.—An increased flow may be temporary or permanent, purely functional (psychic, emotional), or due to local or general organic disease. The ingestion of large quantities of water or such diuretic substances as beer or gin, the use of certain diuretic drugs, hysteria, migraine and humidity with *low* temperature, markedly, though temporarily, increase the amount. *Persistent polyuria* usually indicates actual disease, and is encountered in chorea, pyelitis, amyloid kidney, interstitial nephritis, and diabetes mellitus or insipidus. *Except in diabetes mellitus* and rare cases of interstitial nephritis* *an increased flow results in a lowered specific gravity,* though in diabetes insipidus the *total urine* shows increased total solids.

Persistent and pathologic.

Associated conditions.

Oliguria.—*Pathologic diminution* is usually associated with severe acute congestion, various forms of acute nephritis, chronic parenchymatous nephritis in its active stage, or with circulatory diseases and conditions producing passive congestion. Amongst these are cardiac weakness from any source, cirrhosis of the liver, chronic emphysema, or pressure due to abdominal growths or a pregnant uterus. A marked oliguria accompanies acute infections associated with toxæmia and high body temperature, profuse diarrhœas or hemorrhages, shock or collapse, and in nearly all instances *a reduction in the total quantity of urine is associated with an increase in coloring matter and a heightened specific gravity of the individual specimen.* This last may be present from various causes even though the total excretion of solids is markedly diminished.

Relation to color and weight.

Painful.

Frequency of Micturition and Dysuria.—*Increased frequency associated with pain usually indicates disturbance of the renal pelvis, bladder, prostate or urethra, rather than disease of the kidney itself.* It may

* Ogden reports such an instance—Clinical Examination of the Urine, 1903. The author has never encountered one.

be due to simple concentration and high acidity or more frequently urethritis or cystitis. Relatively or absolutely painless increased frequency may accompany any increased urinary flow, normal or abnormal, or certain stages of active disease, acute or chronic, of either the pelvis or the parenchyma of the kidney. *Complete suppression*, often transient, always serious, may occur either reflexly or in acute or chronic disease of the kidney, and has been known to last for a period of 9 days before a fatal result ensued. *Habitual rising at night* to void urine may be due to urethral stricture, an enlarged or inflamed prostate, gravel, renal or cystic stone, diabetes mellitus or insipidus, malignant or tuberculous diseases of the genito-urinary tract, interstitial nephritis, *insomnia*, and *rarely* habit.

Painless.

Prolonged anuria.

Important sign.

The Color.—Fresh normal urine varies from pale yellow to yellowish red, and all urines may be classified as pale, normal high-colored, or dark. *Deep color is usually associated with relatively high specific gravity and marked acidity; pallor with low specific gravity and lessened acidity, or alkalinity*, diabetes mellitus furnishing an exception to the rule. Indeed so striking is the heavy weight of diabetic urine in connection with its light color, that a provisional diagnosis is often suggested when lifting the tube or bottle.

Relation to color, weight, etc.

Fever Urines.—Are usually high-colored, scant and of high specific gravity.*

Bile.—Greenish, yellowish-green, greenish-brown or deep brown urines may contain bile; if so, the foam produced by shaking the liquid is yellow. Urine containing bile may, when freshly passed, be reddish-brown, but oxidation of the brown bilirubin converts it into biliverdin and produces the greenish tinge.

Brown, Black, Smoky or Red Urines Suggest.—(a). *Blood.* Color red, reddish-brown, smoky, brown, black. (b). *Melanæmia* (melanotic sarcoma). The urine becomes black on standing but *does not* reduce Fehling's solution. (c). *Alcaptonuria.* Urine becomes black on standing *and reduces* Fehling's solution. (d). *Poisoning by coal tar products.* The greenish-black color is due to hydroquinone and follows the excessive use of drugs like carbolic acid, naphthalin, guaiacol, resorcin, salol, creolin and lysol. (e). *Hæmatoporphyrin.* Color of port wine or Bordeaux. **Green urine.**—A green urine results from the administration of methylene blue. **Blue Urine** usually indicates a great excess of indoxyl products, as in ileus, cholera and typhus but

Important data.

* Fever may of course exist in exhausting and wasting diseases with a relatively pale color and low specific gravity.

is rare. **Orange and reddish-brown** urines suggest rhubarb, senna, or chrysophanic acid; **yellow,** santonin.

Milky Urine.—Such an appearance is usually due to pus or chyle. Pus. Pus forms a sediment and is easily detected by the proper chemical Fat. and microscopic tests. *Free* fat upon the surface is almost invariably due to the local use of some unguent, rarely to extreme fatty degeneration Chyle. of the kidney. *Chyluria.* This condition is rare and almost pathognomonic of the Filaria sanguinis hominis. The urine appears like milk and no *fat-droplets* can be detected under the microscope.

Tests for Fats.—The microscopic or macroscopic examination sufficies in ordinary cases. **Chemic Tests.**—Add potassium hydrate and ether, shake, decant and evaporate the supernatant fluid. The fat is taken up, held in solution and remains after evaporation.

Fibrinuria.—In rare instances, as in some cases of villous growth in the bladder, the urine jellies on standing, or, if less fibrin be present, a sticky sediment may adhere to the bottom of the glass.

TRANSPARENCY.—Normal, undecomposed urine should be perfectly clear save for a slight mucous nebula floating in its upper portion, though, even in freshly passed urine, earthy phosphates or the Physiologic sediments. amorphous urates sometimes cause a physiologic sediment, the phosphates being associated with feeble acidity or alkalinity,* the urates with concentration and acidity. *Heating increases the precipitation of phosphates and redissolves urates. Persistent opacity or turbidity is pathologic and may be caused by pus, blood, fat or bacteria.*

ODOR.—The peculiar odor is intensified in sharply acid, concentrated urine and distinctly and characteristically modified in alkaline urine; the volatile alkali (ammonia) being easily detected if present as are indol and skatol in recto-vesical fistulæ, the odor of hydrogen sulphide in hydrothionurea, bitter almonds in nitro-benzol poisoning, etc.

REACTION.—The reaction may be acid, alkaline, neutral or Fixed vs. Volatile alkali. amphoteric, i.e. doubly reactive.† *If alkaline, the physician should determine by repeated tests whether the condition is temporary or permanent* and the alkali *fixed* (potassium or sodium), or *volatile* (ammonia). Any ammoniacal urine to which a few drops of potassium or sodium hydrate solution has been added yields when boiling the odor of ammonia. So also immersed red litmus paper resumes its original color

* Many such urines are really neutral, as shown by a phenolphthalein solution, though turning red litmus paper blue, possibly through the release of CO_2 from the bases.—(F. C. Wood.)

† Presumably because of the co-existence of the *acid* monosodium phosphate and the *alkaline* disodium phosphate.

when dried in the air. A physiologic alkaline or neutral reaction often appears two or three hours after a meal especially in vegetarians, in which case the alkali is fixed and the condition unimportant; but persisent, non-dietetic alkalinity is abnormal and urine *ammoniacal when passed* indicates usually disease of the bladder or prostate. A meat diet tends to produce acidity; a vegetable diet, alkalinity but quantitative determination of the acidity or alkalinity is clinically useless.

"Alkaline tide."

Important facts.

SPECIFIC GRAVITY.—For ordinary purposes a clinical urinometer of the usual type properly graduated for definite temperature (usually 15° C.) is sufficient. In testing freshly passed urines, for every 3° C. above the standard for the individual instrument one point should be added to the right hand figure of the specific gravity. If more accurate results are desired or very small quantities of urine are to be dealt with as in urethral catheterization the hydrometer of Saxe, one of the pycnometers, or, the Westphal balance, may be readily obtained.

Caution.

Technique.—In ordinary clinical work the following precautions are necessary. (a). *The urine must be allowed to cool or proper allowance made.* (b). *All air bubbles must be removed with filter paper.* (c). *The urinometer must be perfectly dry and* (d) *introduced with a little spin to prevent it from adhering to the side of the receptacle.* (e). *The specific gravity should be read from the meniscus or true surface,* the observer's eye being at that level; false readings result if one reads from above, because the fluid rises slightly along the urinometer stem. (f). If *the amount of urine is small, approximate results may be obtained by diluting sufficiently to float the instrument and multiplying the two right hand figures of the specific gravity by 2, 3 or 4 according to the amount of distilled water used.*

Fig. 123.—Simple Urinometer and Accessories.

Factors Determining Specific Gravity.—The normal figures vary from 1.012 and 1.024, the average lying between 1.018 and 1.022. Hysteria and the use of diuretic fluids or drugs may reduce it to 1.002 and in disease it varies from 1.002 to 1.060. In general, *high specific gravity* points to large hemorrhages, profuse perspiration, diarrhœa, fever, and *diabetes mellitus. Low specific gravity* suggests chronic Bright's disease (particularly interstitial nephritis), diabetes inspidus and, hysteria. As a rule, a low specific gravity accompanies increased

Wide variations.

excretion and a high specific gravity, diminished excretion, but there are many exceptions to this rule, many grave diseases being associated with scant urine and deficient elimination of solids, whereas diabetes mellitus may show an enormously increased excretion with the highest readings, and diabetes insipidus an increased amount and low specific gravity *with an actual excess of solids in the 24 hours urine*

Exceptions to rule.

URINARY SOLIDS.—*Normal urine in the healthy adult of average weight should contain about 60 gms. of solids.* Of these about 35 gms. are organic and 25 gms. inorganic. A rough estimation of the amount of solids per 100c c.c. of urine is made by using the co-efficient of Trapp 2.00, or of Häser 2.33 as a multiplier and the last two figures of the specific gravity as the multiplicand: e.g. specific gravity 1.020: $20 \times 2 = 40$ gms. per 1000 c.c. Twenty-four hours urine 1500 c.c. $40 \times 1.5 = 60$ gms. Of this, *about* ½ is urea which should be separately estimated. *The principal inorganic substances* are: HCl (9.35 gms.), phosphoric acid (2.5), sulphuric acid (2.5), nitric acid (1.0), oxalic acid (0.01 to 0.02), sodium (8.0), potassium (3.0), ammonia (0.7), magnesia (0.5), lime (0.3), iron (0.001 to 0.002). *The organic substances* are in gms., urea (30.0), creatinin (1.0), uric acid (0.7), hippuric acid (0.7). Traces of a large number of organic substances including the purin bodies make up the balance for the organic group. *The pathologic substances* are the albumins, blood, bile pigment, bile acids, indoxyl, acetone, diacetic acid, cystin, leucin, tyrosin, carbo-hydrates, phenol, creosol, skatol, urobilin, cholesterin, lecithin, diamins, melanin, fats, fatty acids, lactic acid, beta-oxybutyric acid. Of these indoxyl, acetone, lactic acid, fatty acids, phenol, creosol, skatol, urobilin and even albuminous bodies may be present *in clinically unrecognizable or negligible traces in normal urine.*

Organic vs. Inorganic.

Rough methods.

Note carefully.

Indoxyl (indican).—This normal urinary chromogen results from albuminous putrefaction in the presence of bacteria and if found in the urine in excess suggests intestinal putrefaction, septic processes or the excessive ingestion of red meat. C. E. Simon reports an excess in hypochlorhydria, anachlorhydria *and the hyperchlorhydria of certain gastric ulcers*, and an excess is found in typhoid fever, appendicitis (chronic or acute), cancer of the stomach, peritonitis, chronic gastritis and similar conditions, *reaching its maximum in stasis and ileus. Obstruction in the lower colon or simple constipation does not increase it.*

Significance.

Associated conditions.

Test. (Jaffe-Stokvis).—(a). *Take equal parts of the urine and strong hydrochloric acid.* (b). *Add two or three drops of a saturated solution of sodium or calcium hypochlorite, or of common salt peter.*

Shake. Add 1. c.c of chloroform, shake thoroughly and repeatedly and set aside. The chloroform then shows a depth of color varying with the amount of indican present. Potassium iodide if present may yield an intense carmine and codeine a reddish purple. The observer should establish a normal standard by repeated observations using always the same quantities of urine and reagents. A more accurate test involves the use of a small amount (not an excess) of lead acetate sol. (20%) which removes the urinary pigments in the precipitate.

Quantitative Tests.—Strauss's method. This involves the use of a standard color solution obtained by dissolving one mg. of C. P. indigotin in 1000 c.c. of chloroform. This should be carefully sealed and kept in the dark. Take of urine 20 c.c., add 5 c.c. of lead acetate sol. (20%), which precipitates the urochrome, filter. Mix 10 c.c. of Obermayer's reagent* with 10 c.c. of filtrate (= 8 c.c. urine), add 5 c.c. of chloroform, cork, and shake gently for two minutes. Remove chloroform, add another 5 c.c. of same and so continue until no color is extracted. Of the chloroform used take 2 c.c. in test-tube, add chloroform *guttatim* until the color corresponds with that of the standard solution in a control tube of equal calibre both being held against a white background. The total number of c.c. of chloroform used both in extraction and dilution represents the amount containing 1 mg. of indigo. By multiplying the total amount of chloroform used for extraction by the amount used to dilute to a standard color and dividing by two which corresponds to the amount subjected to dilution we obtain the amount necessary for complete standardization of the total chloroform used.

INDOL (excreted as potassium indoxyl sulphate).—When found in substance in the urine this suggests recto-vesical fistula; otherwise the same statements and test apply as given under indoxyl.

Test of Indol in Substance.—Cholera red reaction. Add to the urine a few drops of dilute sodium nitrite solution and gently pour it down the side of a tube containing sulphuric acid. *Indol yields a purple color at the point of contact,* a diffuse pink on shaking and blue green on neutralization with sodium hydrate.†

SKATOL (excreted as skatol carbonic acid).—It is worth remembering that this substance is especially abundant in cases of tuberculous ulcer-

* Obermayer's reagent. C. P. HCl 1000, ferric chloride 2. This forms a permanent fuming yellow mixture. Accurate quantitative determination is not essential.

† This is simple and sufficiently delicate but other tests such as that of Ehrlich will show 1 part of indol in 400,000 parts of urine.

ation of the intestines, gastric or intestinal carcinoma and pneumonia, and is not increased in the other diseases showing an excess of indoxyl (F. C. Wood). **Rosenbach's Test.**—Add nitric acid, *guttatim*, to *boiling* urine; a *Burgundy red* color with a bluish red foam on shaking indicates skatol, a reddish or brownish red is usually due to urobilin.

UROERYTHRIN.—This normal urinary pigment is increased in febrile conditions, gout, various dyspeptic conditions and after excesses in food or alcoholics. It gives an orange color to the urine and a rose tint to uratic sediments. **Test.**—Add C.P. sulphuric acid guttatim to the urine and a carmine red color reaction appears, or, some pink urates dried or filtered may yield a bright green color with a drop of sodium hydrate solution.

MELANIN.—This brownish black substance produces a dark urine or one darkening upon exposure to the air. This color is intensified by the addition of bromine water or ferric chloride.

UROBILIN.—This or its chromogen is a normal constituent of urine either when freshly passed or as developed by oxidation on standing and is probably the stercobilin of the feces. In excess it indicates an acute infection, lead colic, hepatic cirrhosis, pernicious anæmia, internal hemorrhage, passive congestion as in a weak heart, the ingestion of such substances as antipyrin and antifebrin, chloroform inhalation and the injection of tuberculin (Wood). It may also be the forerunner of, or alternate with, the bilirubin of jaundice constituting urobilin icterus, most frequent in atrophic cirrhosis, carcinoma and pneumonia.

Urobilin icterus.

Test.—Treat 50 c.c. of urine with an equal amount of pure ether, evaporate and dissolve the residue in strong alcohol; the resulting solution should be pale yellow and show a green fluorescence.

Spectroscopic Test.—Dilute urine if necessary, add a few drops of tincture of iodide to each 10 c.c. of urine and the spectroscope will show a band between the green and blue if urobilin itself, not its chromogen, be present.

THE OXYACIDS.—These have the same significance as indoxyl and have no special clinical importance.

ALCAPTONE.—This rare substance has no clinical importance aside from the fact that it produces a urine which darkens on standing and *reduces Fehling's solution*, though not affecting fermentation, phenylhydrazin, bismuth and polariscopic tests.

HYDROCHINON AND PYROCATECHIN.—Both of these substances produce a urine which darkens on standing but are of no clinical importance.

UROCHROME AND HÆMATOPORPHYRIN.—These are constant normal urinary pigments often associated with urobilin and uroerythrin. The first is the chief yellow or amber pigment, the second scant and red. (Urobilin yields yellowish brown and uroerythrin pink.) *Urochrome is readily precipitated by lead acetate solution leaving a clear urine well adapted to spectroscopic examination.* *Hæmatoporphyrin* is often excreted in excess in chronic users of trional, sulphonal, tetronal and the like, the urine being dark brown or black by reflected light. It is probably derived from hematin and may be demonstrated by **Salkowski's Test.**—Treat 30 c.c. of urine with a mixture consisting of equal parts of a solution of barium chloride (10%) and cold saturated solution of barium hydrate, wash the resulting precipitate with water and again with absolute alcohol and shake it up repeatedly with a warm solution representing 10 c.c. of alcohol and 6–8 drops of HCl; a red violet color results and the solution yields the characteristic double bands of acid hæmatoporphyrin.*

Useful procedure.

Creatinin.—This derivative of the ingested meats follows the same laws as urea as to variation in excretion, both in health and disease. It is easily recognized by the *intense* red color produced by adding a little saturated solution of picric acid and a few drops of sodium hydrate (Jaffé's test) to the urine.

Various other substances of no clinical importance but related to the foregoing organic compounds are omitted.

TOTAL NITROGEN.—Before considering urea and uric acid the derivation and importance of the total nitrogen excretion should be considered. While the total nitrogenous output of the human body involves a consideration of the feces, lungs and skin as well as the *urinary nitrogen, the fact that this last represents from 92–93% of the total makes the urinary examination the primary consideration.* The healthy adult on a mixed diet secretes 0.2 gms. of nitrogen per kilo of body weight viz.:—from 10 to 15 gms. of *nitrogen* per day. About 86% of the urinary nitrogen is urea.† 8% is derived from ammonia, creatinin, the purin bodies and the pigments, while 6% represents hippuric acid and unknown substances. The nitrogen excretion measures accurately the variations, not merely of nitrogenous intake, but of body waste and hence the metabolic processes. Increased *by nitrogenous* diet an excess may nevertheless be retained in part for several days, a fact which has not

Factors in excretion.

Ratio to body weight.

Derivation.

Nitrogen retention.

* Narrow dark band in the yellow, a broader one between the yellow and the green in contact with a lighter band in the yellow.

† Though this ratio is not constant.

yet been explained. *Increased excretion* also occurs in acute infectious fevers (excepting acute yellow atrophy of the liver), malignant growths, chronic infections, pernicious anæmia, exophthalmic goitre, scorbutus, the leukæmias, resorption of exudates, diabetes insipidus, phosphorous poisoning and indeed in practically all diseases *associated with marked emaciation, malnutrition or excessive toxic metabolism. Diminished excretion* occurs in nephritis, diseases of the liver and in convalescence from acute diseases, though in the former if associated with marked albuminuria the urine may show an apparent increase if the albumen is not removed. In nephritis especially the stomach, intestines and skin may take on vicarious activity and the feces will show increased nitrogen content. For accurate work, careful measurements of the nitrogen intake and of that of the feces, as well as urinary nitrogen is necessary and the laborious and time consuming process is ill adapted to clinical work. A simpler method is to place the patient upon a milk diet with a known nitrogen content, making daily estimations and then give a rich proteid meal. The promptness of the nitrogenous increase will measure the renal activity and permeability save in certain cases of parenchymatous nephritis, unless the disturbing elements of uræmic diuresis and increased excretion intervenes. For a description of the chemical processes involved the reader is referred to the special works dealing with physiological chemistry.

Not practicable.

Simple device.

UREA.—In the normal individual the urea content varies from 20–30 gms. in 24 hours. *It follows in nearly every particular the laws of total nitrogen excretion already given.* Women excrete less than men, and in vegetarians or poorly fed patients, or those on a milk diet, the amount may not exceed 15 gms. a day. As before stated it represents 86% of the total nitrogen (vegetable diet 79%, rich proteid diet 87%), there are periods of retention lasting several days, and it may be increased by certain drugs such as salicylic acid, caffeine, and quinine. *In diseases of the liver the urea excretion is more greatly diminished than is the total nitrogen*, leucin and tyrosin being coincidently increased, or, in acute processes, an increased ammonia excretion replacing the leucin and tyrosin. In all forms of Bright's disease save the early stage of parenchymatous nephritis urea is diminished and also shows *marked* periodic variations. It is supposed that retained urea in such instances is responsible for the periods of diuresis or diarrhœa so commonly seen. A sudden drop in urea is often noted in nephritis of all kinds and is a danger signal that cannot be disregarded. The author would strongly differ from and indeed, reverse the statement made by certain

Retention periods.

Bright's disease.

Danger signal.

Importance great.

writers that in renal disease urea determinations are less important than casts and albumin.

In several cases presenting violent and intractable neuralgias the attacks were found to coincide in every instance with periods of low urea excretion (6–10 gms. per 1000 c.c.). In others indeterminate but troublesome gastric symptoms, marked neurasthenia and hypochondria were found to coincide with the same condition and in all cases were promptly relieved by treatment thus directed. In several instances no structural disease of the kidney was indicated by the other urinary findings. *The estimation of urea is one of the simplest and most important of all clinical tests.* The most convenient method of quantitative testing is that of Doremus which is sufficiently accurate for clinical purposes.

Value in other conditions.

Note.

Doremus' method.

Description of the Doremus Ureometer.—The Doremus apparatus is much used and consists of a tube carrying a bulb below and so graduated that each division corresponds to 0.001 gm. of urea as represented by the volume of nitrogen evolved at 65° F. There is also a small, curved, nipple-capped pipet holding 1 c.c. of urine. It is filled with Rice's hypobromite solution* to the mark on the long arm of the apparatus, and water added to fill the remainder of the arm and the lower part of the bulb. The pipet† is then filled with urine (*freed from albumin* or *sugar*) up to the c.c. mark, and the point carefully introduced into the bend as far as it will go, while holding the measuring tube perpendicularly, and the contained urine slowly and completely discharged. After the evolution of gas is complete the number of divisions is read off as milligrams per. c.c. or the result multiplied by 100 to obtain the percentage.

URIC ACID.—*Uric acid* (0.2 to 0.1 gm. daily). As a sediment, uric acid is easily recognized by microscopic examination or by the macroscopic deposit of a substance resembling cayenne pepper. The conditions favoring a uric acid deposit are:—(a) *Concentrated urine;* (b) *high acidity;* (c) *deficiency of salts and pigment;* (d) *excess of the uric acid;* (e) *rich proteid diet;* (f) *pathologic increase of metabolism.* The appearance of such a sediment in a single specimen is suggestive of lithæmia and should call for further investigation. The amount of uric acid in *centigrams* contained in each litre of the given

* Sol. I, Caustic Soda 100 and Distilled Water 250. Sol. II, Bromin 1, Potassium Bromide 1, Water 8. To make test solution—Take 5 c.c. of each and to the mixture add 10 c.c. of water. They keep well.

† The instrument can now be obtained with an attached hollow arm with a thumb screw to hold, measure, and release the amount of urine used.

urine may be *approximated* by multiplying the last two numbers of the specific gravity by 2.* Any sediment may be tested for uric acid by heating with a drop of dilute nitric acid upon a porcelain plate; with evaporation a reddish residue appears which strikes a beautiful *deep red* with dilute ammonia. *Quantitative test:*—There is no accurate simple test and the complicated exact tests (Hopkins-Wörner, etc.) are not adapted to the practitioner's use in view of the slight clinical value of the procedure. But one *approximate* method will be given and it, like other quantitative tests, must be applied to a portion of the 24 hours urine to show an excess of uric acid.

Heintz's Method.—To 200 c.c. of clear urine add 10 c.c. of C. P. HCl and mix thoroughly. After 24 hours filter through a dry filter paper *of known weight.* Dry and re-weigh. The difference in weights represents the amount of uric acid in each 200 c.c. of urine.

THE PURIN BASES.—These substances are derived chiefly from the destruction of cell nuclei, hence, from nuclein. Their estimation is difficult and at present, of slight clinical importance.

URINARY CHLORIDES (10 to 15 gms. daily).—**Rough Test.**—Filter the urine if it be not perfectly clear; remove albumin by boiling if necessary (a *trace* does not matter). Prepare a solution of silver nitrate and distilled water (silver, 1 part; distilled water, 8 parts). Add this, drop by drop, to a portion of the suspected urine, *which has been treated with a few drops of nitric acid. A curdy precipitate indicates normal chlorides. Milky turbidity or simple cloudiness shows a marked reduction of the chlorides. No precipitate indicates absence of chlorides.*

Important.

Clinical application.

The value of this procedure in clinical work arises from the fact that *the outpouring of a serous or fibrinous exudate in acute disease is associated with a marked diminution or total absence of the urinary chlorides. One of the first evidences of resolution in lobar pneumonia or of the reabsorption of an exudate or transudate being the reappearance of the lost chlorides.* Their estimation is also of great value in making a differential diagnosis between meningitis and typhoid fever,† they being markedly reduced in the former, and little affected in the latter disease. They are also greatly reduced in acute rheumatism and their disappearance without added joint involvement suggests pericarditis with effusion. In normal individuals a rapid increase of body weight follows the ingestion and retention of the chlorides in quantity, a reduction of weight follows if

* As a matter of fact neither the ratio of uric acid to urea nor to total nitrogen is constant.

† E. S. Wood (Lecture notes).

the substance be withdrawn. Chlorides are more or less diminished in starvation or a milk diet, in febrile diseases generally and by severe vomiting and diarrhœa. *It is now believed that the edema of Bright's disease is largely due to the retention of chlorides in the tissues and a consequent accumulation of water.* This retention is favored by the chemical combination formed by the chlorides, the relative impermeability of the kidney and a poor circulation, hence, it has been suggested that in both incompensated heart disease and nephritis the amount of sodium chloride ingested should be reduced to the minimum.

Relation to edema.

A Simple Quantitative Test (Mohr's) may be used if desired, its errors being so slight as not to affect its clinical value. **Test.**—10 c.c. of urine freed from albumin are diluted wih 100 c.c. of water, a few drops of potassium chromate solution added (enough to produce a yellow color) and the whole placed in a porcelain capsule. This is then titrated with a standard silver nitrate solution ($AgNO_3$ C.P. 29.06 gms. to the litre), 1 c.c. of which should precipitate 1 centigram of sodium chloride; a permanent and diffused orange color ends the titration and multiplying the number of c.c. used by 0.01 gives the amount of chlorides present in 10 c.c. of urine.

The Centrifugal Estimation.—Purdy's method is simple and clinically sufficient. **Test.**—Fill one of the graduated tubes with urine to the 10 c.c. mark, add 15 drops of nitric acid to prevent precipitation of the phosphates (more if the specific gravity be high), add (1–8) nitrate of silver solution, mix thoroughly, and centrifugalize at high speed for 15 minutes; normal bulk percentage reading is from 10–12 and each division represents 0.123 by weight.

THE PHOSPHATES (normal excretion 2.5 to 3 gms.).—Urinary phosphoric acid is met with largely in combination with potassium, sodium, and ammonium ($\frac{2}{3}$), to a less extent with calcium and magnesium. It is derived to some extent from tissue and food nucleins and is increased under a vegetable diet or a *rich* proteid intake. The term phosphaturia is probably a misnomer representing merely a condition of reduced urinary acidity which leads to the constant or almost constant presence of the phosphates as a precipitate. It is met with chiefly in cases of neurasthenia or temporarily in nervous overstrain, and may accompany hyperchlorhydria. Phosphates are readily recognized by the methods mentioned on page 339. Their quantitative determination is of too little importance to be described here. It should be remembered that a phosphatic sediment occurs frequently in normal individuals and many "*phosphaturias*" are little more than the result of morbid

Phosphaturia.

introspection and too careful attention to the appearance of the excreted urine. In ammoniacal urines both the fixed and the volatile alkali combine to form the salts so frequently found in the precipitate.

Two varieties.

THE SULPHATES.—Normal excretion (1.5 to 3 gms.). These exist either as preformed or as ethereal or conjugate sulphates. The former run nearly parallel with urea while the latter follow indoxyl so closely as to justify but slight attention here. These latter are however increased by the ingestion of drugs such as creosol or phenol and phosphorous and occasionally, without an indoxyl excess.

Oxaluria.

THE OXALATES.—*Another condition which modern investigation has shelved is oxaluria*, the diagnosis of which was based chiefly upon the presence of calcium oxalate crystals in quantity in the urinary sediment. These occur usually as a result of gastro-intestinal disturbance to which the symptoms formerly attributed to them are more properly ascribed.

Clinical value slight.

The relation of the crystals to the formation of calculi and their suggestion of disturbed metabolism are the only clinical features of importance. Dunlop has shown that their presence indicates about 25 milligrams of oxalic acid to 1000 c.c. of urine, but this amount does not exceed the maximum normal. Very frequently they are associated with an increase of indoxyl and sometimes with increased ethereal sulphates without increase of indoxyl.

IRON.—This exists in the urine to the amount of 1 milligram but its estimation has no clinical value.

Of slight importance clinically.

AMMONIA (*normal* 0.6 to 1.2).—The proportion of nitrogen derived from urinary ammonia to total nitrogen is about 100 to 5 and the variation of the two substances under dietetic influence is almost parallel. In cirrhosis of the liver ammonia is usually increased as also in hyperchlorhydria, pyloric stenosis and acute gastro-enteritis. It is diminished slightly upon a vegetable diet and the administration of fat not only increases it but causes a corresponding but later increase in the urinary acetone and such urine may show beta-oxy-butyric and diacetic acids. The methods of estimation are too complicated and time consuming for ordinary clinical uses and are therefore omitted.

Albumin in normal urine.

ALBUMINURIA.—*Normal urine does not contain a sufficient amount of serum-albumin to respond to certain clinical tests if these are properly made*, therefore a reaction under such conditions is pathologic. On the other hand *the presence of albumin does not prove a nephritis* as both

Derived albumin.

pus and blood yield a portion of their albumin to the urine and any hemorrhagic or suppurative lesion within or communicating with the urinary tract may cause albuminuria. Again, *the absence of albumin*

in a given specimen does not exclude chronic interstitial nephritis, as in that disease the urine may be albumin free for long periods or appear only at certain times of the day or in definite relation to exertion, mental or physical. *The mere detection of albumin is but a part of the diagnosis* and an estimation of the total urinary solids and urea in the 24 hours urine and recourse to the microscope play a larger part.*

Absence in nephritis.

Relative values.

Albumin in this connection means serum-albumin which is usually associated with serum globulin which acts the same, has essentially the same significance and need not be separately tested. The *urinary mucin* seldom disturbs reactions but *nucleo-albumin* frequently obscures some of the more delicate tests. This substance is increased after over-exertion, in actual inflammation of the urinary tract, especially in purulent accumulations, in leukæmia, jaundice and acute infections with marked toxæmia. **Test.**—*Nucleo-albumin* is readily detected by diluting the urine with three times its bulk of water and rendering it strongly acid with acetic acid.

Varieties.

Mucin.

Nucleo-albumin.

ALBUMOSES chiefly the secondary appear in the urine and have some clinical importance because of their relation to suppurative processes. Their presence, for example, in a pleurisy would suggest empyema; in meningitis, a septic rather than a tuberculous process; in a severe intestinal lesion, dysentery or typhoid rather than tuberculous ulceration; in hepatic disease, abscess or suppurative cholecystitis; in tumors, malignant ulceration. **Test.**—Secondary albumoses are precipitated by acetic acid saturated with sodium chloride or the biuret *reaction* may be obtained as follows:—Remove all albumin by treating the urine with sodium acetate and then with ferric chloride until it produces a deep red color, neutralize carefully with sodium hydrate, boil and filter, repeat if a potassium ferro-cyanide test applied to the fluid shows albumin. Nucleo-albumin and mucin may also need to be first removed if present in quantity. This is readily accomplished by strongly acidulating with acetic acid and filtering after several hours. 50 c.c. of albumin free urine are then acidulated with HCl (5 c.c.) and phosphotungstic acid sol. added until no further precipitate is obtained, gentle heat is then applied until the precipitate shrinks and collects on the bottom of the receptacle. After decanting, the precipitate is repeatedly washed, dilute sodium hydrate

Indicate suppuration.

* One of the commonest sources of error arises from contamination of the specimen by leucorrhœal discharge or the pus and blood of urethritis, less commonly there are fistulous openings, draining abscesses of the surrounding structures.

added and the solution warmed until the deep blue becomes a light yellow. After cooling a dilute solution of copper sulphate is run down the side of the tube or beaker and a *rose color* indicates albumose.

The Bence-Jones' Proteid.—This was formerly supposed to be an albumose but is as yet a doubtful body. It appears in cases of osteomalacia and multiple myeloma and constitutes one of the clinical curiosities. Its most characteristic feature is its behavior when heat is applied to urine containing it. Commencing as an opacity at 50° C. becomes precipitated at about 58° C., and increases up to 70° C. when it begins to disappear and at boiling point has returned to its first opalescence. Without discussing the theories relating to the causation of albuminuria one may say that under certain conditions any or all of the proteid bodies found in blood plasma may appear in the urine.

A clinical curiosity.

The Significance of Albuminuria.—Osler aptly covers the whole field in saying "the presence of albumin in the urine in any form and under any circumstances may be regarded as indicative of changes in the renal or glomerular epithelium, a change, however, which may be transient, slight and unimportant, depending upon the variations in the circulation or upon irritating substances taken with the food, or, temporarily present, as in febrile states." We know that albuminuria may follow *emotional disturbances*, violent exercise, cold baths, the ingestion of excessive amounts of nitrogenous foods or infections of various kinds. In young persons it may be irregularly intermittent or occur at definite intervals (cyclical), wholly lacking the general and circulatory symptoms of Bright's disease.

Changes in epithelium.

Functional albuminuria.

Tests for Albumin.—As a result of an extended series of tests undertaken by Dr. P. A. Hoff and the author covering both normal and abnormal urines he is convinced that for the general practitioner the only safe and reliable tests are *the heat-and-nitric acid procedure and the heat test with acetic acid and brine.* Every other test used in his experiments gave confusing results when it became a question of determining the small traces of albumin yielding a haze but no definite precipitate. The nitric acid layer test is also practical and accurate *if performed with care and a proper knowledge of its disturbing factors.* In this connection several other tests are mentioned because of their general acceptance by physicians but they are not recommended.

Best tests.

General Considerations.—The essentials of a clinical test for albumin are (1). *Simplicity.* (2). *A reasonable degree of delicacy.* (3). *Decisiveness. Simplicity* reduces careless or imperfect work to a minimum; if one exceeds a reasonable *delicacy* he finds disturbing reactions in

almost every urine, and finally, a test must be so *decisive* as to make unnecessary the more complicated tests for disturbing urinary proteids and permit a positive and convincing opinion. *The heat-and-nitric acid test as usually applied does not fulfil these requirements.* Merely boiling the urine coagulates both serum-albumin and nucleo-albumin, and precipitates the phosphates. Nitric acid alone coagulates serum-albumin, the primary and secondary albumoses, nucleo-albumin and the resins, and precipitates urates, if present in excess. The careless application of the layer test (Heller's test) may result in the confusion of the low-lying or rather, junction point band of albumin with the higher stratum of nucleo-albumin or resins, or, the albumin ring may be obscured in dark colored urines by the pigment developed at the point of contact. *Another, but unpardonable source of error lies in the failure to hold the tube toward the light but against a black background such as the coat sleeve or a book.*

Faulty method.

Disturbing factors.

The Proper Application of the Heat-and-nitric Acid.—Clear the specimen by filtration or if bacterial (*persistent* cloudiness is usually due to bacteria) by adding barium carbonate, shaking andfiltering.

HEAT-AND-NITRIC ACID TEST.—If the urine be concentrated, dilute it with from two to three times its bulk of water; if of low specific gravity, add $\frac{1}{5}$ its volume of a saturated solution of sodium chloride. Then (a) fill two *perfectly clear* test-tubes $\frac{2}{3}$ full of the urine and *set one aside* for subsequent comparison of transparency. (b). *Boil the upper portion* of the liquid in the remaining tube and directly *after* boiling add 1 or 2 drops of strong nitric acid and 1 or 2 drops more if no *persistent* cloudiness or precipitation appears. (c). Any such *persistent cloudiness or precipitate* is *serum-albumin.* Caution. *Do not add acid before boiling nor boil after the addition of the acid.** Resins may be present and cause opacity if turpentine, oil of sandalwood, copaiba and the like are taken and bile pigment may precipitate in icteric urines, but both disappear on shaking up with alcohol.

The test.

THE NITRIC ACID CONTACT TEST (Heller's test).—Place in a conic glass or test-tube a dram or two of pure nitric acid, and, using a pipet, allow the urine to flow gently down the side of the inclined glass and upon the surface. If the mouth of the pipet be placed against the side of the glass, *just above the level of the acid,* and a very gentle flow established, the result is extremely clean-cut and

An excellent method.

*The latter procedure especially will often cause the conversion of large amounts of albumin into a soluble acid albumin as is readily proven by any one interested.

beautiful. *Albumin, if present, appears as a band or ring at the junction of the two fluids* and is more or less distinct in proportion to the amount of albumin present. The tube or glass should be set aside as several minutes may be required to develop the reaction. *Urinary coloring matters appear near the surface of the acid, but usually at a point just below the albumin ring*, if the test has been properly made. *If within ten minutes there appears another ring resembling albumin, but at a distinctly higher level, one may assume that a relative excess of uric acid or acid urates is present, or, that the individual from whom the urine was received has fever.* *

Site of band.

Time required.

Inferences.

Mucin and resins.

Mucin, if present in excess, may form a light cloud in the upper portion of the urine, and *resins* may also be precipitated but are known by their immediate disappearance when a portion of the cloudy urine is withdrawn by means of a pipet and shaken with alcohol. If space permitted, much that is of interest might be said of the urinary chromogens and pigments. If one wishes to study them the urine should be placed in the glass and a considerable quantity of the acid poured rather briskly down the side. When this is done, the coloring matters are prominently displayed and the albumin ring, if present, is raised to a level that nearly corresponds to that of uric acid and acid urates, when the test is applied by the first method.

Useful manœuvre.

Relative value of tests.

The heat-and-nitric acid and the cold nitric acid tests as thus applied are excellent and quite sufficient for ordinary purposes. *Of the two, the heat-and-nitric acid test is the more definite and positive. On the other hand, the so-called "heat-and-nitric acid test" as generally used is fallible and misleading.* It will be remembered that this method consisted in adding nitric acid, drop by drop, *while boiling the urine.* The following errors may then occur:—*A small amount of albumin treated with an excess of acid may form a soluble acid albumin and escape detection*, on the other hand, *in an alkaline or neutral urine with phosphates present in excess, a failure to acidify the specimen may result in the formation of a soluble alkali albuminate* and *neither acid nor alkali albuminates are precipitated by subsequent boiling.* Furthermore, according to Purdy, mucin, globulin and albumoses are precipitated by this method. The author recently saw

Faulty method.

Confusing reactions.

* According to C. E. Simon the subsequent appearance of a film-like hoar frost on the sides of the glass indicates that about 25 gm. of urea are contained in a litre of the urine under examination. Similarly spangles of this urea nitrate point to 45 gm., and, the separation of a dense mass to 50 or more gms. to the litre. It must be remembered that such deductions are not of much value unless the specimen be a part of the 24 hours urine.

a case in which a specimen of urine containing 0.25% of albumin was reported normal after the application of this test.

THE ACETIC ACID, HEAT AND BRINE TEST.—First Step.—To a portion of the urine submitted for examination that will two-thirds fill two test-tubes add two or three (never more) drops of acetic acid (50 per cent.). **Second.**—*Filter* the amount necessary for the test *until absolutely clear*. A single filtration usually suffices; if not, the cloudiness is probably due to bacteria, which pass readily through ordinary filter paper. They may be easily removed by adding barium carbonate, shaking, and filtering. **Third.**—Set aside a portion of the previously acidified *filtered* urine for comparison. **Fourth.**—Add to the remaining urine one-sixth of its volume of a saturated aqueous solution of common salt and fill a test-tube two-thirds full. **Fifth.**—Boil the upper portion thoroughly and compare with the unheated specimen, holding both to the light against a black background, as previously recommended. Any precipitate or cloudiness is due to serum-albumin. The amount of time ordinarily involved is trifling, and the test is extremely definite and delicate. A fine test.

The only reagents required are: (*a*) acetic acid (50 per cent.); (*b*) a saturated solution of common salt, *but the procedure must be carried out in the exact order here given.* Be exact.

The Potassium Ferrocyanide Test.—Into a clean test-tube pour a dram or two of acetic acid (50%), to this add twice its volume of an aqueous solution of potassium ferrocyanide (1: 20). Shake the mixture, and overlay with the suspected urine, as is done in the nitric acid contact test. Albumin, if present, appears at once as a band at the junction of the two fluids. *The full amount of acetic acid must be added.**

Robert's Test Solution (HnO_3 1 part, saturated solution of magnesium sulphate, 5 parts) may be used as in Heller's test but is if anything too delicate and subject to the same elements of error. **Spiegler's Test.**—Is too delicate for clinical work showing one part of albumin in 250,000 of urine containing abundant chlorides. **The tri-chloracetic acid test** can be made by dropping a crystal into the urine contained in a small test-tube. It is extremely delicate, partially precipitates albumose and, in concentrated urines, urates as a distinct *upper* ring. These three may show albumin in all urines, or so nearly so, as to rob the finding of a *trace* of any clinical significance.†

* *Disadvantages.* It precipitates albumoses and nucleo albumin.

†Nevertheless the author has seldom found a *distinct ring* with tri-chloracetic acid without confirmatory microscopic findings.

Simple tests.

QUANTITATIVE TESTS FOR ALBUMIN.—Two simple tests suffice for the quantitative estimation of albumin. The first demands the use of *Esbach's albuminometer;* the second requires *a centrifuge.* Both tests are extremely simple in execution and as no physician's office is complete without a centrifuge and as the Esbach tube is inexpensive and easily obtained, no hardship is involved in their application. Esbach's albuminometer is merely a glass tube graduated and lettered as shown below. This tube is filled to the letter U with urine, if necessary previously diluted until its specific gravity is 1008 or lower, and a solution of picric acid (picric acid, 10; crystalline citric acid, 20; distilled water, 1000) is added until the level of the liquid has reached the letter R. A rubber stopper is then inserted, the tube is inverted several times to thoroughly mix the urine and test solution and is then set aside for from 24 to 48 hours. The albumin is precipitated and its height, measured by the numerals upon the scale, represents in gms. the amount of albumin present in one litre (1000 c.c.) of the urine. This method is sufficiently accurate for practical purposes (although peptones, mucins, etc., if present, are precipitated together with the albumin), but is slow, and far inferior to Purdy's direct centrifugal method.

Esbach's method.

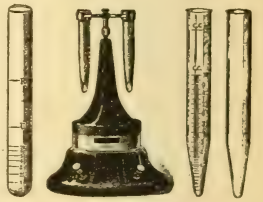


Fig. 124. Fig. 125.

Fig. 124.—Esbach's albuminometer.

Fig. 125.—Electric centrifuge.

Centrifugal Method.—Fill the graduated tube supplied with the centrifuge to the mark 10 c.c. Add 3 c.c. of a 10% solution of potassium ferrocyanide and 2 c c. of acetic acid (50%). Mix thoroughly, set aside for ten minutes, and, *thoroughly revolve in the centrifuge until the supernatant fluid is clear and the albumin evenly deposited* at the bottom of the tube. Each mark represents $\frac{1}{10}$ c.c. and corresponds to a bulk measure of 1% or $\frac{1}{60}$ of 1% by weight (Odgen).* The old fashioned methods of estimation by boiling the urine in a test-tube, or by judging the amount by the depth of the ring produced by the contact test with nitric acid are useful, but not sufficiently accurate and introduce too much of the personal equation. On the other hand, the very exact methods of the chemical laboratory are laborious, and demand more time than the busy practitioner can give. The follow-

Best clinical method.

* If urates are present in excess the supernatant fluid may be replaced by boiling water after centrifugalization and the process continued a short time longer.

ing classification is employed by E. S. Wood and Ogden for rough work and is based upon the cold nitric acid test performed as follows:—An ordinary wine glass is half filled with urine, inclined until the liquid nearly overflows and then underlaid with nitric acid, poured in as slowly as possible, until it equals one-third of the urine volume. If then the wine glass be perfectly clean and placed obliquely in front of a dark background, the observer facing the light, they distinguish (1) the self explanatory "*faintest possible trace.*" (2). *A very slight trace*, i.e. a faint cloud only definitely seen against the dark background, (3) *a trace*, visible without background, both from the side and from above but not concealing the bottom of the glass when so viewed, (4) *A large trace*, i.e. a sharply defined zone but not flocculent nor wholly opaque when viewed from above. (Represents about 1-10 of 1%.) (5) *A $\frac{1}{8}$ of one per cent.* reaction, i.e. a sharp non-flocculent zone obscuring the bottom of the glass, (6) $\frac{1}{4}$ of 1%, band flocculent and opaque, (7) $\frac{1}{2}$ of 1%, band dense and flocculent.*

Wood's method.

Rough estimation.

PUS.—Pus in the urine is readily recognized by chemic or *microscopic* tests (see page 342). As a gross sediment *phosphates* and the *pale urates* may mislead the careless observer, but should never cause confusion. Urates are dissolved by heating. Phosphates disappear if an acid be added, whereas both the foregoing procedures will increase a turbidity due to pus. **Chemic Test.**—Add liquor potassæ to the suspected urine and shake the solution vigorously. The persistence of suspended air-bubbles and viscidity indicate pus. If the amount be very large, or if the liquid potassæ be added to the sediment after decanting the supernatant fluid, a gelatinous mass results. The microscopic test is more definite when mere traces of pus are present (see page 342). The addition of a drop of *very dilute* acetic acid solution to a microscopic preparation clearly brings out the nuclei of pus cells under the microscope.

Simple and valuable.

BLOOD (hæmaturia, hæmoglobinuria).—The color of urine containing blood in quantity has already been described. In general it may be said that acid urines containing *considerable* blood are dark or smoky, and alkaline urines bright red. Oftentimes its presence must be detected even though the amount is insufficient to color the urine, and moreover mere color cannot be depended upon in any case. For the detection of small quantities the microscopic examination, chemical

Color deceptive

*It will be seen that such distinctions require much practice but from personal experience in the Harvard laboratory the author can attest the accuracy there attained.

Smoky urines.

tests and the spectroscope are extremely important as the coloring matter alone may be present primarily or as the result of fermentation changes. The color due to poisoning by coal tar products closely simulates the smoky urine of hæmaturia.

Heller's Test for Blood.—(a). Boil the urine in a test-tube. (b). Add caustic soda and continue the boiling as long as precipitation continues. *If blood be present, the phosphatic precipitate is brownish-red and the supernatant fluid, a bottle-green.* This test is said to detect 1 part of blood to 1000 parts of urine.*

The Guaiacum Test for Blood.—Shake in a test-tube equal parts of old turpentine (or hydrogen peroxide solution) and fresh tincture of guaiacum. Pour this mixture gently down along the side of the tube so as to overlay the urine. If *blood or pus* be present a blue band appears at the point of junction. If this be due solely to blood, it persists when the temperature of the mixture is raised to the boiling-point, whereas, if pus alone be present, the color disappears. As oftentimes both are present in the urine, the reaction may lack precision.†

Blood vs. Pus.

The Sources of Hemorrhage.—Aside from acute Bright's disease or severe acute congestion the commonest sources of blood are urinary calculi and new growths. Clots occasionally exist if the blood is from the bladder, or small clots may represent the lumen of the ureters. Blood from the *kidney* is usually well distributed and lacks clots, and further indications of its source are found in the presence of blood casts and fibrinous casts. If from the *kidney pelvis*, the blood cells are thoroughly distributed and associated with the peculiar epithelium of that region. It may be due to calculi, acute inflammation or varicosities, and associated pus points to inflammation whether simple, septic or tuberculous. *Bladder* hemorrhages suggest ulceration, calculi, tumors or acute inflammation and, rarely, such parasites as the Filaria sanguinis hominis may be found with a chyluria. If from the *urethra* the first jet of urine contains the blood or pus, though if from the *prostatic* portion it may appear *chiefly* at the end of micturition. *Accidental blood* may occur especially in the female and must be excluded. In hæmoglobinuria or methemoglobinuria the blood cells are scant or absent and the spectroscope shows the characteristic bands of oxyhæmoglobin, reduced hæmoglobin or methemoglobin. This condition

Clots.

Localizing features.

Hæmoglobinuria.

* If the urine be alkaline and its phosphates precipitated one may add some acid normal urine before testing.

† If the slightest trace of copper remains from Haines' or Fehling's test in an imperfectly cleaned tube a false reaction will appear.

occurs in poisoning by nitro-benzol, sulphonal, antipyrin, phenacetine, naphthol, mushrooms, in severe tropical malaria, and certain cases of syphilis, typhus and scarlatina.

BILE.—Bile acids and bile pigment are both found in the urine under certain conditions. *Bile pigment*, if present in considerable quantity is readily detected by the yellow-tinted foam produced by shaking. *The best simple chemic test is made as follows:*—Filter the suspected urine several times through the same filter paper. Drop *fuming* nitric acid upon the wet paper and watch for the characteristic color-play, viz., orange, red, violet and green, the last being the essential color. Rhombic crystals of bilirubin are readily obtained by shaking up thoroughly with chloroform, decanting and evaporating the chloroform extract. **Marechalt's Test.**—Overlay a portion of the suspected urine in a test-tube with an alcoholic solution of iodin (tincture iodin, 10 parts; alcohol, 90 parts). If bile be present, a beautiful green band appears at the junction point.

Simple tests.

Haycraft's Test for Bile Acids.—Is made by dropping a pinch of "flour of sulphur" upon the surface of the suspected urine; if the *bile acids* are present the powder drops to the bottom of the tube. This old test has been recently studied by Frankel and Cluzet. They find it accurate, sensitive, and as applied to the urine, very definite.* The reaction depends upon the disturbance of surface tension and the presence of bile acids indicates the presence of hepatogenous jaundice, though their absence does not exclude it.

A good test.

TESTS FOR GLUCOSE IN URINE.—All clinical tests for glucose in solution depend upon the following properties:—(a). *The fact that when brought into contact with certain oxids, as for example, those of copper and bismuth, it becomes oxidized at their expense*, i.e., acts as a reducing agent. (b). *Its ready fermentability.* (c). *The fact that it is dextrorotatory.* (See also p. 337. Lactose, pentose, etc.)

The Copper Tests.—The well-known *Trommer's test* has been superceded by better and more accurate methods, but *Fehling's* solution is so widely used as to require a description of the test. *This solution is distinctly inferior to Haines' modification and is so unstable that it must be kept in two parts and mixed whenever needed for use.*

Relative value.

Fehling's Test.—*Directions.* (a). *Remove albumin by boiling and subsequent filtration.* (b). *Pour into a test-tube one finger's breadth each*

* From personal experience with a large number of "unknowns" a certain number of which contained bile, the author is convinced that this is a good clinical test.

Test fluid.

of the following stock solutions, which when mixed should form a *deep blue solution.*

Stock solutions.

Solution A.—Dissolve 34.64 gm. of pure, dry, powdered copper sulphate in 200 c.c. of warm distilled water and add distilled water to make 500 c.c. of the *light blue solution.* **Solution B.**—Dissolve in 300 c.c. of hot water 180 gm. of Rochelle salt. Filter. Add of pure caustic soda, 70 gm. Cool, and add distilled water enough to make 500 c.c. of a *colorless solution.* Keep in a dark place.

Heat test solution to boiling point, add at once 20–30 drops of the suspected urine and *boil no longer*, but in the absence of a reaction set aside for from 5–30 minutes, and try the polariscopic and fermentation tests, a positive reaction *proves* nothing but the presence of a reducing agent unless the ultimate precipitate is *red*, not yellow or green.

Objections to Fehling's Solution.—Fehling's solution as ordinarily prepared is open to serious objections:—(a). It is unstable. (b). An excess of glucose obscures the terminal reaction by becoming caramelized if boiling is prolonged. (c). It cannot be directly applied to ammoniacal urine unless such be especially prepared. (d). A large number of substances may reduce its cupric oxid. Such are glycuronic and glycosuric acid, alkapton, creatinin, uric acid, and various drugs such as benzoic acid, chloroform, chloral, glycerin, the salicylates,

Real value.

turpentine, etc. Hence, if one uses Fehling's solution for qualitative work, he must bear in mind, that it is more valuable as a negative than as a positive test. *A urine that does not reduce Fehling's solution is free from glucose, but reduction does not conclusively establish its presence.*

A better test.

Haines' Solution.—A much simpler and absolutely permanent copper solution is that of Haines.* **Application of Test.**—Boil a few c.c. of the solution gently in a test-tube, add guttatim 6–8 drops of the urine, boil *gently for a moment only.* The reaction is identical with that of Fehling's solution.

For albuminous urines.

Allen's Test.—This is said by Hutchison and Rainy to have the following advantages:—(a). Albumin need not be removed. (b). Uric acid, creatinin, and like substances do not affect the reaction.

The test derives its value from the fact that acid solutions of sodium acetate precipitate the interfering substances without removing or affecting any glucose that may be present.

Test.—In a perfectly clean test-tube heat 8 c.c. of urine to the boiling

* Formula: Take of pure copper sulphate 30 grains, distilled water 1 ounce. Make a perfect solution and add of pure glycerine ½ ounce, mix thoroughly, add liquor potassæ 5 ounces.

point. Pay no attention to any precipitate (albumin). Add 5 c.c. of the solution of copper sulphate used in making Fehling's solution (solution A, as described). Cool partly. Add 2 c.c. of a saturated solution of sodium acetate that has been rendered faintly acid by acetic acid. Interfering substances are now precipitated. Filter and add to the clear filtrate, 5 c.c. of the solution B used in making Fehling's solution. Boil twenty seconds. If sugar is present, the solution becomes opaque and green, and deposits, either immediately or after standing a few minutes, a *fine yellow precipitate.* The tests already given are placed in the direct order of their convenience and rapidity, but the inverse order of accuracy. Yet error will seldom occur if every sugar reaction be checked by the one accurate test for glucose, namely:

Absolute test.

THE FERMENTATION TEST.—This test depends upon the fact that *glucose is a fermentable substance, and practically the only one that urine ever contains.* (See p. 337.)

The Test.—The most convenient apparatus is that of Einhorn. *Each step in the process should be checked by comparison with a normal urine;* therefore two tubes are required. If Einhorn's saccharometer is not to be had, the Doremus urometer tubes will answer the purpose. (a). *Boil the two specimens for several minutes,* to drive off any air they may contain. (b). *Add to each a pinch of tartaric acid in order to maintain their acidity* and prevent ammoniacal decomposition. (c). Dissolve in each the same amount of German yeast (about one-sixth of a fresh yeast cake is sufficient). If there be any doubt as to the freshness of the yeast, it is well to test it with a solution of glucose. The long limb of the fermentation tube of the saccharometer or of the Doremus ureometer is filled with urine, and the two specimens are placed side by side in a warm place. About 24 hours are required for complete fermentation and quantitative estimation, but the diagnosis can often be made in a very short time, as the appearance of any considerable amount of gas proves the presence of glucose. *By using Einhorn's saccharometer the amount of sugar may be determined with clinical accuracy by the scale measuring the volume of gas produced.*

Simple apparatus.

Technique.

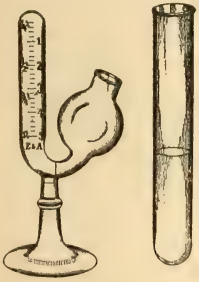


Fig. 126.—Einhorn's saccharometer.

Rougher Method.—Lacking any form of fermentation tube, the test may be made by taking the specific gravity of two specimens, to one of which yeast is added, and both then are set aside in a loosely

Specific gravity method.

stoppered flask or bottle and in a warm place. If glucose be present fermentation occurs the specific gravity is lowered and each degree of density lost roughly corresponds to one grain of glucose to the ounce of urine (0.21 gm. to 100 c.c.). *The specific gravity should not be taken the second time until the urine has cooled to the temperature it had when the first estimate was made.*

Caution.

Quantitative Estimation with Whitney's Reagent.—In addition to the previously described tests may be mentioned that of Whitney, which is more rapid than the fermentation test, but very inaccurate and depends upon the decolorization of an ammonio-cupric-sulphate solution.

Not recommended.

Test.—One dram is heated to boiling point and the urine is added drop by drop, the mixture being boiled for from three to five seconds after each addition. If no change occurs, the process is continued until ten or fifteen drops have been added. If sugar be present, the blue color begins to fade, and is finally entirely removed, leaving a colorless solution. The amount of sugar present is then estimated by the following table:—

If reduced by Minims.	It contains to the Ounce.	Percentage.
1	16 or more grains	3.33
2	8 grains	1.67
3	5.33 "	1.11
4	4 "	0.83
5	3.20 "	0.67
6	2 67 "	0.56
7	2.29 "	0.48
8	2 "	0.42
9	1.78 "	0.37
10	1.60 "	0.33

If the amount of sugar be large, as indicated by the loss of color following the addition of one drop, the urine should be diluted by doubling or trebling its volume by the addition of distilled water, and the result then obtained must be multiplied by two or three as the case may be.

The duration of the boiling period must be neither more than five nor less than three seconds. In the author's hands it has proven far inferior to the fermentation test.

The Polarimeter.—Direct estimation of the sugars by this means is by far the most satisfactory method, requires no special training but

the instrument is somewhat expensive. The percentage of sugar is read directly from the vernier scale in the clinical instruments such as Ultzmann's but it is usually best to decolorize the specimen with lead acetate solution; the urine must be albumin free and the containing tube dry or if containing water, it should be rinsed out with some of the urine to be tested. Technique.

LEVULOSE.—Levulose is abundant in ripe fruits and honey, is absorbed unchanged and occasionally appears in the urine, yet it can be taken in quantity in diabetes without causing a marked excretion and in ordinary glycosuria may replace cane sugar or glucose in the diet. Instances of pure levulose diabetes have been reported but they are exceedingly rare. If 100 gms. of honey be taken on a fasting stomach by a non-diabetic not more than 10 or 15% will show levulosuria but in *diseases of the liver such as cirrhosis, carcinoma and syphilis the same test results in a marked excretion and indeed the substance may appear spontaneously in such cases.* **Test.**—The saccharometer (polarimeter) should show rotation to the left, *but the substance reacts to fermentation and Fehling's solution* exactly like glucose. For identification therefore one must use **Seliwanoff's Test.**—Add to 10 c.c. of urine (showing lævo-rotation) a little resorcin and 2 c.c. of dilute HCl; *a bright red reaction* appears on heating if levulose be present. Important data. Clinical value. Misleading reactions. Necessary test.

LACTOSE appears in the urine of women after childbirth, not in pregnancy and more frequently in those who do not nurse the child (80% as against 20%). It is most abundant about the fifth month after delivery and seldom exceeds 0.5%. **Test.**—*It reduces Fehling's solution* and is fermentable with difficulty but whenever any sugar reaction appears in women after delivery **Rubner's Test** should be applied:—To 10 c.c. of urine are added 3 gms. of lead acetate, the resulting precipitate is filtered off and the filtrate heated until it assumes a brownish color, and again after the addition of ammonia. The appearance of a *brick red color and a cherry red precipitate indicates lactose* if there be more than 0.3%. Specimens of high specific gravity should be diluted one-half and less amounts require evaporation of the urine and a test of the residue. Lactation glycosuria. Specific test.

PENTOSE.—After the ingestion of large quantities of fruit, or more rarely after taking excessive amounts of vegetables, coffee, tea or milk, pentose *may appear in sufficient amount to reduce Fehling's solution.* A very few cases of true pentosuria with otherwise unimportant symptoms have been observed. *It does not yield gas production by fermentation* which differentiates it from the more important substance, glucose. Fermentation absent.

MALTOSE.—Its chief interest lies in its presence in certain cases of pancreatic disease but the tests are not suitable for the practitioner.

Not fermentable.

GLYCURONIC ACID.—This substance may in certain combinations *reduce Fehling's solution*, but it has no definite clinical significance, *does not undergo fermentation with yeast and is levorotatory in acid solution*. The only definite test is that of Newburg, a description of which may be found in the physiological chemistries.

ACETONE, DIACETIC ACID AND OXYBUTYRIC ACID.—One need only consider the two former substances clinically, though it is probable that they are derived from oxybutyric acid by oxidation. A distinction may be drawn between the diabetes of a severe and that of a mild type by the amount of acetone and the behavior of the acetone and diacetic acid as affected by diet. They are prone to appear if the diet is rigidly proteid and disappear when a mixed diet is allowed *but if they persist in quantity the case is a bad one and the end not far distant.* The significance of acetone and diacetic acid is essentially the same for both, but the former may precede the latter and should be sought in diabetic urines if diacetic is absent, *the latter being first tested for*, because of the simplicity of its reactions and because it is invariably pathological though not confined to diabetes.

Important in prognosis.

DIACETIC ACID.—Test.—If a few drops of the tincture of ferric chloride be added to the urine in the presence of diacetic acid a bordeaux red appears together with a precipitate of phosphates. For accurate results these should be allowed to settle or be filtered out and the addition continued.* The urine must be fresh or the test is of no value.

Simple test.

ACETONE.—Legal's Test.—Distill a few c.c. of urine by using a simple distillation flask with its neck corked and its lateral arm leading into a test-tube or ordinary flask; a bunsen burner or alcohol lamp completing the outfit. The distillate collected in three or four minutes is treated with a few drops of a fresh solution of sodium nitro-prusside, a few drops of acetic acid are added and the mixture rendered alkaline by sodium hydrate. If acetone be present a red appears deepening to carmine and purple red. **Lieben's Test.**—Add to distillate a few drops of Lugol's solution and sodium hydrate. If acetone is present it will form a macroscopic or microscopic sediment of the star like yellow crystals of iodoform and on heating the characteristic odor may appear.

Two good tests.

Simpler method.

THE PRESERVATION OF SPECIMENS FOR EXAMINA-

* The disappearance or marked diminution of the color on heating suggests the presence of salicylates or coal tar products which may yield a similar reaction.

TION.—Urine must be examined before decomposition or the findings are worth little. One need only have the patient add one or two thymol crystals, an ounce of cold saturated solution of boracic acid, or salicylic acid gr. xxx, to the quart of urine to ensure its preservation. Formalin is less desirable as it forms a precipitate.

THE EXAMINATION OF URINARY SEDIMENTS.—The accurate microscopic examination and correct interpretation of urinary sediments demand a thorough knowledge of the special technique involved. Gross error is possible in two directions. 1. *Through failure to detect important abnormal elements.* 2. *Through misinterpretation of elements found.* Every physician should not only own a microscope, but be able to use it, and no time can be more profitably employed than that which is spent in acquiring a correct technique. He, who graduates from the schools of to-day, is or should be trained to a degree that will enable him to do reasonably accurate work in this line. On the other hand, many excellent men of the older generation through lack of early training and opportunity are sadly deficient in this important branch of clinical medicine.

Illumination.—In examining a urinary sediment microscopically, the first essentials are *correct focusing and a comparatively dim light.* Every microscope should be provided with an iris diaphragm and a nose-piece holding three objectives, one low power, one medium, and for the third, a high power oil-immersion lens. In urinary work only the first two are required, save in those cases in which the tubercle bacillus is the object sought. The physician should commence operations with a dim light and his low-power lens (1 inch or ½ inch objective), this being by far the best for finding casts as it distinctly shows their outline and gives a larger field. He should then slightly increase his light and bring his medium power objective (¼ to ⅛ inch) into focus. This brings out the structure of casts and renders distinct any cellular elements or crystals that may be present in the field. Having once found any object of interest the lenses and the illumination may be varied at will.

Light and lenses.

Substances found in Urinary Sediments.—The following are the substances most frequently found in urinary sediments:—*Extraneous Material* such as fibers of cotton, linen, wool or silk, various vegetable forms from the rinsing water, if it be not distilled, starch-grains, etc. *Phosphates.* The macroscopic appearance of the phosphatic deposit is well known. The grayish-white material so often mistaken for pus is composed of amorphous calcium and magnesium phosphates and their

ready solubility in mineral acids at once identifies them. They are found only in alkaline urine, and, if ammoniacal fermentation has occurred, will be associated with beautiful crystals of the triple phosphate. Calcium phosphate may also appear in crystalline form.

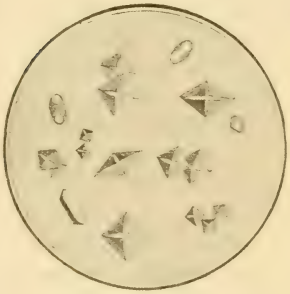


Fig. 127.
Calcium oxalate crystals.

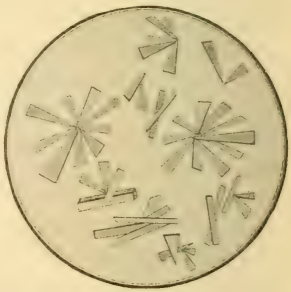


Fig. 128.—(*After Jakob.*) *a*. Calcium phosphate. *b*. Calcium sulphate.

Urates. The ordinary deposit of urates occurs in moderately acid, concentrated urine during the process of cooling, or when exposed to an unusual degree of cold. The color varies from yellow to rose-red, the latter constituting the so-called brick dust deposit (sedimentum lateritium). Upon application of heat they promptly disappear. When

Fig. 129.—Ammonio-magnesium (Triple) phosphate.

Fig. 130.—Uric acid crystals.

nitric acid is added to a urine rich in urates, a deposit of nitrate of urea is formed. The deposited urates are amorphous, excepting only the ammonium urate, which occurs in ammoniacal urine as the so-called thorn-apple crystals (see fig. 131).

Uric Acid.—Uric acid may be precipitated from any urine, if concentrated, during the so-called acid fermentation or in hot weather when the high temperature prevents precipitation of the urates. Excessive acidity and concentration or a pathologic excess may occur in certain

Significance of deposit.

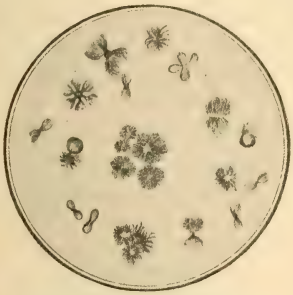


Fig. 131.—Ammonium urate.

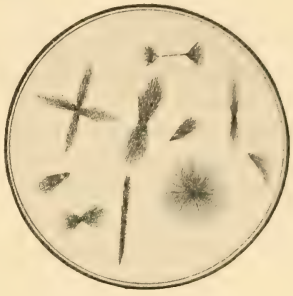


Fig. 132.—Tyrosin.

conditions, but, *when passed,* urine should never contain the crystals as a precipitate. The macroscopic deposit resembles cayenne pepper. The microscopic appearance is best shown by the plate (see fig. 130).

Calcium Oxalate.—This rarely forms a visible sediment and is abnormal if found in urine freshly passed. The crystals are character-

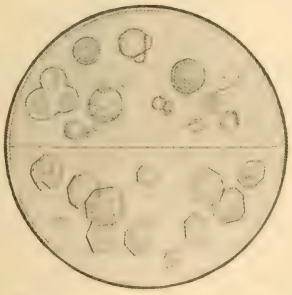


Fig. 133.—*a.* Cystin. *b.* Leucin.

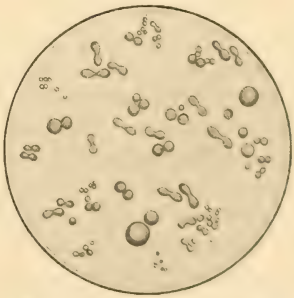


Fig. 134.—Calcium carbonate. (*After Jakob.*)

istic and unmistakable (see fig. 127). Leucin, tyrosin, and cystin are rarely seen, but may be readily recognized by comparison with the illustrations (see figs. 132, 133).

Calcium Carbonate.—This is occasionally precipitated with the

earthy phosphates. Usually it is amorphous, but occasionally forms crystals shaped like a dumb-bell. It is easily recognized by the effervescence produced when a mineral acid is added.

The Organized Sediment.—The organized constituents of the urinary sediments are far more important than the inorganic. The chief elements or substances are:—(a). *Blood.* (b). *Pus.* (c). *Spermatozoa.* (d). *Bacteria.* (e). *Epithelium.* (f). *Casts* (*pseudocasts, true casts*). Rarely one may find parasites or their ova and fragments of new growths.

Blood.—The blood as it appears in the urine may be quite normal in appearance, or on the other hand be so changed as to make its recognition difficult. If hemorrhage has taken place in the urethra, prostate, bladder, ureters or in the pelvis of the kidney, and the urine is acid and fresh, the red corpuscles appear as yellow biconcave discs, with rounded edges and a light central portion. If, on the other hand, the hemorrhage has taken place into the cortical portion of the kidney and the urine has stood for some hours or is alkaline, the corpuscles may become *pale and swollen*, their diameter lessened and they may appear as *mere shadowy circles* and as such are easily overlooked. *Crenation* of the normal cells may also occur in a urine that is deficient in salt. Such cells have irregular, star-like processes along their border, but retain the yellow tinge of the normal cell.

Erythrocytes.

Phantom cells.

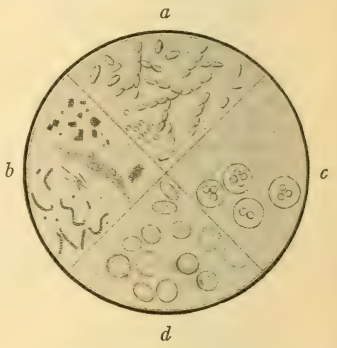


Fig. 135.—*a*, *b*. Various forms of fungi and bacteria. *c*. Pus-cells before and after treatment with acetic acid. *d*. Various forms of red blood-cells.

Pus.—Pus in quantity is in most cases easily detected by the chemic test. The pus cell as seen under the microscope is precisely like the white cell seen in a smear preparation of normal blood, but is less easily recognized as such in the urine. In the acid urine the pus cells are *usually larger than the red cells, are colorless, granular and as a rule have several nuclei readily distinguished by careful focusing.* The presence of these nuclei serves to distinguish them positively from the red cells. Any doubt upon this point may be readily removed by allowing a drop of very dilute solution of acetic acid to run beneath the cover-glass, assisting the process, if necessary, by laying the edge

Leucocytes.

Valuable device.

of a piece of filter or blotting paper against the opposite edge. The acetic acid dissolves the granules and brings out clearly the cell nuclei, *but unless very dilute, will destroy any hyaline casts that may be present.* In ammoniacal urine, the pus corpuscles are soon destroyed, becoming agglutinated and losing their structural characteristics. Caution.

Varying Significance of Pus in the Urine.—As to the significance of pus in the urine, it may be remembered that (a). *Pus that comes with the first jet of urine, the remainder being clear,* is from the urethra. (b). *That a moderate amount of pus occurring in an acid urine is usually from the renal pelvis;* but may be due to tuberculosis of the bladder. (c). *Large quantities of greenish pus point to rupture of an abscess* into the urinary passages or to a pyelonephrosis.* (d). *Pus in an ammoniacal urine is usually from the bladder.* In all cases the diagnosis must depend upon the character of the associated epithelium and the presence of casts or specific micro-organisms. Important data. Note.

Spermatozoa are easily recognized by their well known form (see fig. 145).

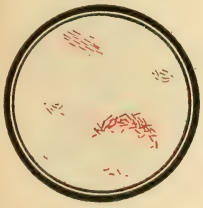


Fig. 136.—Tubercle bacilli in urine. Observe tendency to form groups.

Bacteriuria.—Aside from the tubercle bacillus, many forms of bacteria may be found in the undecomposed urine, and their presence may be unattended by symptoms or accompanied by a variable amount of irritation of the bladder. So, also, in fermenting urine one finds both bacteria and spores. The spores are highly refractile and tend to form chains (see fig. 135). The most important bacterium is the tubercle bacillus, only to be recognized by such staining methods as are described on page 348. *The ordinary bacterial urine is persistently cloudy, has a musty odor, and presents on shaking a swirling appearance, as if fine grains of sand were put in motion.* Yeasts, etc. Macroscopic test.

Epithelium.—In spite of the efforts put forth by microscopists, *the exact differences existing between the epithelial cells of different portions of the urinary tract have been only in part determined.* Such variations are shown by the plates, but must be learned from the careful study of specimens obtained from known cases of cystitis, nephritis, etc., a mere description being of slight value. The *renal cell* as seen in the urine is usually small, round and mononuclear. *Cells from the straight tubules* are somewhat larger and have a more irregular shape, being sometimes square or polygonal, but *all renal cells are mononuclear.* Limits of differentiation.

* Most commonly prostatic.

Clinical symptoms must control findings.

Caudate cells arranged in overlapping layers are often described as the characteristic cells of the renal pelvis, but as a matter of fact, they represent only the superficial layer, and are seen only in early or mild cases of pyelitis. *The cell from the deeper layer strongly resembles the renal cells. Ureteral cells* are spindle shaped. *Bladder epithelium* is generally of the well known tesselated or pavement variety, and may be distinguished from *vaginal epithelium* only by the fact that the cells are somewhat smaller, never overlap, and occur in a single layer. *Vaginal epithelium* is rather larger; the cells overlap like the shingles on a roof, and are likely to occur in masses consisting of several layers. The *epithelium from the neck of the bladder, prostate, and the calices* of the kidney pelvis are almost identical.

Fatty Renal Cells and Compound Granule Cells.—The former are much smaller than the latter which are large, round, bulging with

Fig. 137.—*a*. Epithelium from renal pelvis. *b*. Vaginal epithelium.

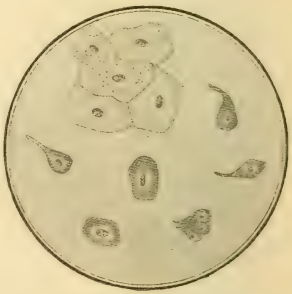


Fig. 138.—Bladder epithelium. (*Various forms.*)

refractile granules and frequently spiculated by fatty crystals. They have no exclusive disease relation but may be found in any form of renal lesion characterized by fatty casts and in other chronic inflammatory and ulcerative lesions of the urinary tract as a whole. Renal cells reflect the changes shown by the various types of casts.

CASTS.—Casts are of two kinds. (1). *The true cast, which has its origin in an exudate from the tubules of the kidney.* (2). *The pseudo-casts, or cast-like bodies, which may or may not originate in the kidney.*

Doubtful origin.

True Casts. *The exact nature and source of the true casts are not positively known.* Whether they consist of disintegrated and modified epithelium, of a morbid secretion of the epithelium, or are simply coagulated materials exudate from the blood, *the fact remains that they appear*

clinically as casts of the renal tubules, having to a great extent a form and calibre corresponding to that portion from which they come, and appearing hyaline, waxy, fibrinous, granular, fatty, epithelial or bloody, according to the nature and extent of the underlying pathologic changes. If we assume that their basis is that pathologic change known as hyaline degeneration, it becomes very easy to understand most of their modifications. Such a process would lead ordinarily to the formation of a *hyaline cast.* If the granules of the degenerated cell adhered to or became intermixed with the deposit a *granular cast* would result, and this would be finely or coarsely granular, light or dark, according to the activity of the pathologic process. Fatty degeneration in the cells would be reflected in the *fatty cast.* Adherent desquamated cells would form *epithelial cast,* adherent blood cells, the *blood cast,* etc. It is reasonable to suppose that the basis is the same for all varieties, and that

Important fact.

A convenient hypothesis.

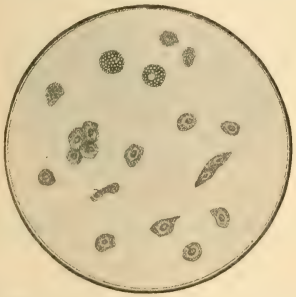


Fig. 139.—Various forms of renal cells, including "compound granule cells."

Fig. 140.—*a.* Blood cast and hyaline cast carrying blood-cells. *b.* Leucocyte or pus-cast. *c.* Hyaline cast carrying renal cells. *d.* Epithelial casts.

in the hyaline, waxy, and fibrinous casts we have but slight variations in structure, though the conditions attending their presence may be quite different and distinct, and their significance definite and important. *The hyaline cast is transparent, and shows an apparently homogeneous structure; its size is variable, its ends rounded, and its sides are nearly parallel.* Its shape is best shown in fig. 141. Unless careful focusing and proper shading of the light be employed it will certainly escape detection (see fig. 142). Representing, as these hyaline casts do, the least degree of pathologic or at least of inflammatory change, it becomes at times a difficult question to determine the significance and importance of an *occasional cast.* Some observers report them as present in nearly every urine if the centrifuge be employed but such

Significance of hyaline casts.

has not been the author's experience. Occurring alone and unassociated with albuminuria or other symptoms pointing to diseased kidneys, it must be admitted that their significance is less grave than would have been assigned them by the older teaching. It is certainly true that they may represent transient and hence often negligible conditions or localized rather than general renal changes but as a *persistent* constituent of the urinary sediment they still demand serious consideration and have lost little of their old significance. It is also a much debated question as to whether they are ever found without some degree of albuminuria and according to E. S. Wood, the hyaline cast

True casts seldom trivial.

Fig. 141.

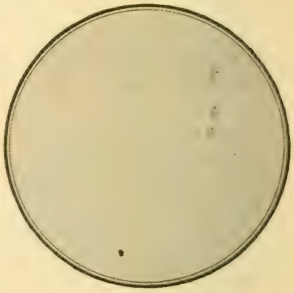


Fig. 142.

Figures 141 and 142 show the effect of low and high illumination. In figure 141 hyaline casts are plainly visible, one carrying four renal cells. In figure 142 *the casts are entirely lost* in the flood of light, and only the renal cells appear.

Hyaline and granular.

is always associated with the pale very finely granular cast.* It rapidly disintegrates in alkaline urine or in that treated with acetic acid. *The chief significance of the hyaline and finely granular cast,* existing alone, *is found in their predominance in chronic passive congestion and interstitial nephritis.*

The Waxy Cast.—*This is highly refractile and usually of large size. It stands out as clearly upon the field as does the triple phosphate crystal,* Significance. *and once seen, is never forgotten.* It points to one of three conditions: *amyloid kidney, advanced chronic parenchymatous nephritis and the terminal stage of interstitial nephritis.* Ultzmann states that it may be Prognostic value. present in tuberculosis of the kidney. In *amyloid kidney,* it appears *earlier* than in *interstitial nephritis.* Indeed, *in the latter it marks the beginning of the end* and cases of contracted kidney in which it is found

* This corresponds to the author's own observations as applying to ordinary clinical work and to *true* casts and seems to have a logical pathologic foundation.

will usually terminate fatally within one year. **The Fibrinous Cast.**—This misnamed cylinder resembles the waxy cast save in color. It is always yellow or even brown, deriving its color from blood pigment, and, as might be expected, *points to an active or subsiding inflammation*, ordinarily to acute Bright's disease. **The Blood Cast.**—This *points to acute inflammation, as does the brown* (dark) *granular cast.** *The coarse granular cast* is usually associated with fibrinous or with waxy casts. **The fatty cast** is most frequently seen in *convalescent cases of acute nephritis, or* in *chronic parenchymatous nephritis*. **Epithelial casts** occur only in *acute* or *subacute inflammation, in acute Bright's*

Color important.

Important inferences.

Fig. 143.—Granular cast light and dark, coarse and finely granular.

Fig. 144.—*a*. Waxy casts. *b*. Fatty or fat-bearing casts.

disease, or the subacute exacerbation of a chronic parenchymatous nephritis. Many casts carry here and there an epithelial cell, yet cannot properly be called epithelial casts. **Pus casts** are found in suppurative disease of the kidney or in pyelitis extending to the straight tubules and frequently require treatment with dilute acetic acid to distinguish them from epithelial casts.

Significance and Association of Casts.—*Hyaline and Finely Granular Casts indicate:*—(a). Interstitial nephritis. (b). Amyloid kidney. (c). Chronic congestion of kidney. *Waxy Casts indicate:*—(a). Terminal stage of chronic interstitial nephritis. (b). Advanced parenchymatous nephritis. (c). Amyloid kidney. In *Acute and Subacute Nephritis one finds* (a). Fibrinous, (b). Blood, (c). Epithelial, (d). Hyaline and (e). Dark granular casts. *In convalescence from acute nephritis one finds* fatty, dark granular, epithelial and hyaline casts.

* If, as is usually the case, the cast carries washed out cells (abnormal blood) it indicates slow effusion of blood or a high origin—if normal blood, more abundant hemorrhage or an origin in the straight tubules or pyramids.

In *uncomplicated chronic parenchymatous nephritis one finds*, fatty, hyaline and finely granular casts and with any decided disturbance epithelial casts.

Sources of Error in Searching for Casts.—*Cast-like Forms.* The amorphous urates are frequently grouped in cast-like forms and similar accumulations of bacteria and other sedimentary substances may perplex the tyro. They will seldom or never deceive the trained eye as they lack the definite boundary-line of the genuine cast. Urates, moreover, are promptly dissolved by heating the preparation. More deceptive still are the *cylindroids*. They are usually longer and more band-like than the hyaline cast, and more likely to be convoluted or twisted; usually they take the form known as mucous cylindroids which occur in many non-albuminous urines and cannot be mistaken for true casts as they appear flattened and ribbon like, often with frayed ends and faint longitudinal striation. The author believes that fewer casts would be reported in otherwise normal urines *if the observer regarded all doubtful forms as disproven* until he could find in the same urine a definite cast of the ordinary size and form and with both ends rounded. *If of renal origin the unusual will certainly be accompanied by the common form.* In a case recently observed periods of hypochondria were accompanied by *showers* of spurious casts without the slightest change in the chemical constitution of the urine.

Accidental casts.

Misleading forms.

Mucous cylindroids.

Demand typical forms.

Cylindroid storms.

Fig. 145.—Spermatozoa and associated substances in urinary deposit. *a, a, a, a.* Spermatozoa. *c, c.* Spermatozoa, tail out of focus. *d, d, d.* Amyloid corpuscles. *e.* Prostatic cast. *g.* Crystals. *h.* Lecithin-granule cells. *k, k, k.* Epithelium.

Prostatic Plugs.—These may closely resemble the large hyaline or fibrinous cast but usually carry or are associated with spermatozoa, and are associated with evidence of prostatic irritation or inflammation.

The Examination of the Urine for Tubercle Bacilli.—*Tubercle bacilli may be found in urines that are in every other respect almost normal.* If such a condition be suspected, the urine must be subjected to prolonged treatment in the centrifugal machine. The sediment is then examined precisely as in the case of the sputum (see page 114), save that a small quantity of egg albumin is added to the specimen before placing it on the cover-slip.

Pyuria sometimes absent.

THE GONOCOCCUS.—As indicating the infectivity of any persistent secretion after urethritis the presence of gonococci is important.

Method.—The usual smear is made, dried in the air and fixed, and indeed may be made by the patient if no discharge is present save in the early morning or at irregular times. Ordinarily the Jenner's stain or the alkaline methylene blue are sufficient. In any case of unusual importance it may be necessary to use cultural methods as it is claimed that the micrococcus catarrhalis may cause confusion. The so-called *urethral thread*, though common after gonorrhœal infection, *indicates no more nor less than chronic congestion of the deep urethra.*

Simple method.

Shreds.

EHRLICH'S TYPHOID DIAZO-REACTION.—The test was introduced by Ehrlich in 1882, and, after meeting much opposition and criticism born of misunderstanding and misapplication, has at last been accepted at its true value in most of the important clinics.

Solutions Required.—*Solution A.* Hydrochloric acid, 50; distilled water, 1000; sulphanilic acid, q.s.ad sat. (should be thoroughly saturated). *Solution B.* Sodium nitrite (not nitrate), 0.5 per cent (½ of 1%), solution in distilled water. *Solution C.* (Test Solution.) *One hundred* parts of A plus one part of B.

The original test solution of Ehrlich was made by adding to 40 parts of Solution A 1 part of Solution B. This the author has slightly modified by using 100 parts of A to 1 of B, with the effect of eliminating many disturbing factors and doubtful reactions. Dr. C. E. Simon suggested the ring method of testing described below. *To apply the test, take equal parts of C and the urine. Shake thoroughly and add aqua ammonia in excess, allowing it to run gently down the tube so as to overlay the mixture below.* If the reaction be present, *a deep-red band* appears at the line where the ammonia meets the mixture; when shaken, it yields a *pink or rose-colored foam* and after standing several hours, a green precipitate forms. The following rules must be carefully observed:—(1). *Use fresh urine.* (2). *See that the reaction is acid and the urine filtered.* (3). *Use a fresh test-solution.* (4). *Keep the sodium nitrite solution in a black bottle, and in a cool place and renew it frequently.* The sulphanilic solution keeps indefinitely. (5). *Hold the tube near, but not against, a white background, the source of light being behind the observer. Artificial light should not be used.* (6). *Accept no color but a distinct red, and regard no reaction as a true one in which the solution when shaken does not yield a pink foam. Pseudo-reactions* occur in which the band is of the proper color, but the foam is yellow or brown. The most absurd errors have arisen from a failure to observe

Author's modification.

Technique.

Important rules.

Absurd errors.

Sources of error.

the exact technique here outlined and some excellent men have used sodium *nitrate*, weak ammonia, or even omitted the addition of ammonia altogether, and naturally failed to get consistent results. Others have admitted yellow and orange reactions, or have even used a 5% solution of sodium nitrite. The test is not pathognomonic, as was originally maintained by Ehrlich, but is constant in all severe forms of typhoid, appearing sometimes as early as the fourth or fifth day, *though more generally at the end of the first week or ten days*, and persisting *until the fever begins to decline*. If the test be applied according to the author's method the *true* reaction is absent in malaria, appendicitis, pneumonia and the earlier stages, at least, of acute miliary tuberculosis, occurring only in the author's experience in some of the exanthemata, in certain cases of advanced malignant disease, late in miliary tuberculosis and in febrile cases associated with *septic absorption*. Pseudo-reactions are found in a considerable number of diseases and the inclusion of yellow and orange reactions has discredited a good test. It is of great value, as a negative sign, for it is the author's firm belief, *that it will be found present in all severe cases of typhoid, and that its persistent absence in any such case quite certainly negatives the diagnosis of typhoid.**

Real value great.

Pseudo-reactions.

URÆMIA.—Any form of Bright's disease may be associated with symptoms grouped under the head of "uræmia." The exact cause of this condition is unknown but its occurrence depends upon the retention of certain excretion products distinctly toxic in their nature. It is a well known fact, that *no living organism can resist the poisonous effect of its own waste products*, and in the elimination of these substances the kidney plays the chief part. The symptoms of uræmia are made manifest chiefly through the *nervous system*, although the gastro-intestinal and respiratory tracts are almost invariably involved.

Cerebral symptoms.

Nervous Symptoms of Uræmia.—*Almost every disease of the nervous system may be simulated by uræmia*, but the cerebral symptoms are the most interesting and important. Headache, drowsiness, stupor or coma may be found individually or as transition stages. There may be slight twitching or the more terrible convulsive seizures closely simulating epilepsy, and violent outbreaks of acute mania, profound melancholia, or the so-called delusional insanity of Bright's disease may easily lead to serious error in diagnosis or even to the committal of such patients

* Recently Unverricht, in quoting Michaelis an enthusiastic advocate, has raised anew the old question of color and demands a more definite reaction. A *deep red band and a pink foam* may and *should* be demanded by every clinician before the individual reaction is accepted.

to an asylum for the insane.* Various *disturbances* of *general sensation* may be encountered such as itching, anæsthesia, hyperæsthesia, formication and abnormal response to heat and cold. *Vertigo* is often a prominent feature and is usually associated with periods of marked circulatory depression, diminution of total solids or the predominance of the dyspeptic symptoms, often so prominent a feature in this disease. The most remarkable symptoms of uræmia are those extraordinary attacks of *paraplegia* or *hemiplegia* simulating true apoplexy, and the *disturbance of the special senses* resulting in uræmic tinnitus aurium, deafness or sudden temporary blindness (uræmic amaurosis).† *Various more serious eye symptoms* may co-exist with uræmia *and the diagnosis of chronic interstitial nephritis is often first made by the oculist*, who discovers evidence of a neuro-retinitis of the albuminuric type. Uræmic amaurosis usually lasts but one or two days and ordinarily follows some profound manifestation of uræmia, such as coma or convulsions. *Cases presenting marked symptoms of actual albuminuric neuro-retinitis seldom live longer than one year.*

Sensory.

Paralyses.

Eye symptoms.

Respiratory Symptoms.—Cases of chronic nephritis are often peculiarly subject to inflammation of the pulmonary structures, but aside from these we have curious *disturbances* of *respiratory rhythm* and uræmic dyspnœa may be continuous, paroxysmal, alternating, or Cheyne-Stokes. *The paroxysmal type is often mistaken for true asthma*, the mode and time of onset being identical. *Continuous dyspnœa* of uræmic origin is not uncommon, and medical literature furnishes many evidences of an *ambulatory Cheyne-Stokes type.*‡

Dyspnœa.

Gastro-intestinal Symptoms.—*These so closely simulate various independent diseases as to render diagnosis impossible, except by reference to the urine and particularly the estimation of urinary solids.* As pre-

Urinary solids.

* A remarkable case in the author's practice showed the existence of a uræmic delusion, single but fixed and dominant, which lasted about three years, during which period the patient was converted from the warmest friendship to the most bitter enmity, returning suddenly to his first status at the end of the period. In this case the albuminuria was intermittent with frequent periods of marked deficiency of excretion, as indicated by estimation of the urinary solids. Several physicians pronounced the man wholly free from disease, because of the absence of albumin from individual specimens of urine.

† Amaurosis, i. e., blindness usually temporary and removable and without apparent lesion may also be encountered in hysteria, migraine, acute severe hemorrhages, tobacco and cocaine habituation and diabetes.

‡ In one case of interstitial nephritis in the author's practice, typical Cheyne-Stokes breathing was present at night for over three years before the patient's death and was occasionally present in the daytime.

Uræmia sine nephritis.

viously stated, these uræmic symptoms may be evident in any case of nephritis; the author believes that they may also be present in minor degrees at least without actual nephritis, if the functional activity or capacity of the kidney is temporarily deranged or lost. Nevertheless its most extreme manifestations are encountered in chronic interstitial nephritis, or even more commonly in chronic diffuse nephritis *with exudation.** *The onset of uræmia is almost invariably associated with lessened quantity of urine and a sharp reduction in total solids and urea.*

Note.

Uræmic Coma.—This may closely simulate apoplexy, but is rarely so sudden in onset and is more generally preceded by convulsions, a history of which should be carefully sought. Such cases are the *bête noir* of the hospital physician on account of the difficulties in differentiation. The following points should be noted:—(a). *An examination of the urine obtained by catheterization is of the first importance*, as it may yield evidence of an active or chronic nephritis, or show a marked reduction in solids. (b). A *history of antecedent convulsions* or convulsive movements may be obtainable. (c). *The pupils yield no certain signs.* (d). *Paralyses are rare* though there may be muscular twitching and rigidity of the extremities if severe convulsions have occurred. (e). The temperature may be elevated, but usually it is normal or subnormal. (f). *Ophthalmoscopic examination may show retinal changes.* (g). *The general aspect of the patient may clearly indicate the existence of renal disease.* (h). *Little dependence can be placed upon the odor of the breath* as indicating renal toxæmia. Uræmic odor is sweet, nauseating, chloroform-like and may pervade a whole apartment, but is found in connection with profound toxæmia, resulting from cardiac failure, advanced hepatic disease, renal disease, and sometimes in connection with malignant growths. It lacks the peculiar quality of the breath as encountered in diabetic coma in which there is an actual fruity fragrance of the most penetrating quality.

Essential data.

Uræmia vs. Aceto-næmia.

CRYOSCOPY.—The introduction of cryoscopy by Korányi represents an effort to secure more exact information especially concerning renal activity and efficiency by determining the freezing point of the blood and urine. The greater the molecular concentration of a watery fluid the lower is its freezing point and assuming that the average freezing point of normal blood ranges from —0.56° to —0.58° C. and that of the urine

* A case under the author's observation for five years showed during that period nearly every symptom to which reference has been made in a preceding page, and amongst these were attacks closely simulating hysteria, but promptly removed by radical therapeutic measures, while no less than three times before death the patient was found in deep uræmic coma.

between —0.9° and —2° C. it is evident that the lower freezing point on the part of the blood indicates a greater concentration and that a higher figure for the urine indicates a lessened molecular content. It further appears that a test of the urine taken by catheterization from each kidney might determine the relative functional activity of those organs and that the procedure would prove a guide to the surgeon in nephrectomy as well as to the physician in connection with the so-called uræmic states and renal inefficiency.

THE CRYOSCOPE.—The Fontaine instrument* consists of a freezing chamber containing a properly supported central tube within which yet another tube may be so adjusted as to leave an air chamber between them. Within the latter is a delicate thermometer registering from—3° to 3° C. in graduations of 1-200 and this is encircled by a spiral stirrer, neither touching the sides of the innermost tube. **Method.**—The large tube being in position the freezing chamber is filled with cracked ice and rock salt in alternate layers terminating in an upper layer of salt an inch in depth; 10 c.c. of the blood or urine have in the meantime been placed within the inner test-tube and put in or upon ice to cool during the preparation of the freezing mixture (5 to 10 minutes). The small tube is then placed within the large one and fitted with the thermometer, care being taken that the latter's bulb is immersed but that the instrument is at no point in contact with the wall of the inner tube. After about 5 minutes of continued stirring a slow fall followed by a rapid drop occurs to about —2° C. at which point the reading is stationary for some minutes and then rises to the *true freezing point*, again falling to the temperature of the freezing mixture. The reading should be made with the eyes at the level of the mercury column. It is assumed that a freezing point of —0.6° C. or lower for blood and 1° C. or higher for urine indicates a sufficient degree of renal inefficiency to forbid surgical interference. As might be expected this method has its enthusiastic advocates and no less strenuous opponents. Space will not permit a full discussion of the strength or weakness of the procedure, but a just idea may be obtained by reading Sahli's admirable, critical discussion,† and the book of its enthusiastic advocates, Casper and Richter.‡ Such careful observers and enthusiastic workers as DaCosta and Cabot are unwilling to subscribe to the claims made for it, and to the author it appears abso-

*Made by G. Fontaine, 16 Rue Monsieur la Prince, Paris, or the highly recommended modification of the A. H. Thomas Co., Philadelphia.

†"Diagnostic Methods."

‡"Functional Diagnosis of Kidney Disease."

lutely impracticable as a working clinical procedure, and one so subject to serious error from slight causes and to wide variation in apparently normal cases as to cast grave doubt upon the findings obtained.

RENAL INEFFICIENCY.—(Renal inadequacy.) Aside from cryoscopy many efforts have been made to determine the functional efficiency of the kidneys, especially with relation to actual or suspected medical or surgical diseases of those organs, but so far no method has been evolved which is free from serious objection and at times gross errors. Indeed it would seem at present that the excretion of total solids as represented by the specific gravity figures, and the special procedure for the determination of urea and the chlorides offer the best means available for the busy practitioner. So far as the personal experience of the author is concerned these have proven adequate and seldom misleading in medical work and to them no objection can be urged which cannot be met by an equal or greater counter objection to the proposed substitutes. The estimation of total nitrogen can hardly be adapted to the examination of specimens obtained by ureteral catheterization which represent small separate specimens, and furthermore involves an absolute knowledge of the ingesta, of excretion through channels other than the kidney, a complicated chemical estimation, and repeated tests covering a considerable period; all of which requirements *place it entirely outside the possibilities of clinical work under ordinary conditions.*

Simple methods.

Clinically adequate.

Total nitrogen.

Methylene Blue.—Achard and Castaigne some years ago recommended and used methylene blue for the determination of the functional activity of the kidneys, using intramuscular injections and noting the time elapsing between its administration and the subsequent discoloration of the urine as well as the approximate amount of the coloring matter recovered. It has been found, however, that the test is of little value as a measure of renal insufficiency, the elimination being unmodified in parenchymatous and amyloid nephritis, thus proving that important renal constituents do not act as does the dye.

Obsolete.

The Relative Toxicity of Urines.—Bouchard's method of determining the relative toxicity by injecting urine into guinea pigs is so full of error variations as to be entitled to little respect, nor is it in any way adapted to ordinary clinical work.

The Phloridzin Test.—The objections to phloridzin as a test substance are much the same as against methylene blue, though it is probable that an estimation of the glycosuria following the administration of this drug furnishes an *approximate* idea of the functional capacity of the kidney.

Of some value.

Comment.—It would seem to the author that in view of the fact that no unobjectionable or infallible method has been evolved, one is justified in falling back upon those that are simplest and that furnish clinical results of decided significance. In other words, *until better methods of an accurate and decisive nature and greater simplicity are evolved, the practitioner is justified in depending upon the rough estimation of total solids and the approximate determination of nitrogen by the urea content.* As a matter of fact in those conditions associated with uræmic crises there is seldom any doubt as to the temporary or permanent disability of the kidneys, nor, *as before stated is this confined exclusively to renal disease*, though the urine gives the best indication. Careful observation of a number of cases *presenting the symptoms of a modified uræmia but with apparently normal kidneys, and of precisely similar cases with known renal disease* has convinced him that the *decided reduction in urea, preceding, and associated with the onset in each and every one of these cases, together with the disappearance of all symptoms under treatment directed to a restoration of renal activity* strongly suggests the possibility of a *purely functional inefficiency*, associated with retention symptomatically indistinguishable from the uræmia of Bright's disease. Furthermore in all these cases not only was the specific gravity decreased, but urea was in every case *less than half the normal*, and, although the diet was uniform, cessation of symptoms and the rise in the urea percentage and specific gravity were invariably coincident.

CHRONIC PASSIVE CONGESTION OF THE KIDNEY.—This is always venous and secondary to diseases of the heart, lungs or liver, or to pressure on renal veins by abdominal tumors including the pregnant uterus and ascitic fluid. **Morbid Anatomy.**—The kidneys ordinarily show a simple venous hyperæmia. Secondary and venous.

Symptoms.—There are no symptoms of importance save those referable to the primary lesions and the urinary findings. **Urine.**—The total amount is diminished, total solids approximately or actually normal. *Reaction.*—Acid. *Color:*—Distinctly high save in diseases characterized by marked impairment of nutrition. *Albumin* is present only as a trace except in pregnancy when large amounts may be found. **Microscopic Findings.**—An occasional hyaline or finely granular cast, together with a few renal cells.

The Kidney of Pregnancy.—Even though the condition may be one of simple pressure hyperæmia, the amount of albumin is likely to be larger than in other instances of chronic passive congestion, and it must not be forgotten that active hyperæmia or acute or chronic inflammatory

diseases of the kidneys may accompany this state. In this connection it is of the utmost importance that specimens of urine be submitted frequently to the attending physician and that these represent the 24 hours urine, and not the individual hap-hazard specimen. Furthermore, the essential feature of the examination, as indicating danger to the patient, lies in the estimation of urinary solids, particularly of urea. This point is often overlooked by the practitioner, who may mistakenly regard the amount of albumin as the important feature.

Caution.

Total solids.

ACUTE CONGESTION OF THE KIDNEY AND ACUTE NEPHRITIS.—These conditions should be considered together, inasmuch as the causes operating to produce them are essentially identical, and further, because no line can be drawn between actual acute nephritis and severe congestion, save that based upon the duration of symptoms. Mild congestion of the kidney of the arterial type occurs under many conditions of slight potency, and is the antithesis of the severer types, the amount of urine being increased, but a small trace of albumin being present, and the urinary solids being actually or approximately normal.

Mild vs. Severe congestion.

Etiologic Factors.—Any acute congestion and inflammmation. *Irritating Drugs.*—Among these are copaiba, cubebs, sandal-wood oil, turpentine, cantharides, carbolic acid, phenol compounds generally, arsenic, lead and mercury. These cause trouble only when ingested in excessive doses, or for too long a period. *Insoluble Urinary Constituents.*—Uric acid, calcium oxalate, cystin, acid urates and phosphates are the chief factors. *Toxines.*—These may be divided into three groups:—(1). Those associated with acute febrile infections and especially with the exanthemata and certain virulent tropical diseases of which yellow fever is the chief. (2). Suppuration with septic absorption. (3). Chronic diseases such as syphilis, gout, malaria, pulmonary tuberculosis, diabetes mellitus and certain of the anæmias. *Nervous Influences.*—It has been customary to place under this head the nephritis attending such conditions as acute mania, delirium tremens and the so-called ascending or reflex congestions connected with diseases of the bladder, seminal vesicles, urethra and the prostate. It is probable that this is incorrect. In many of the cases of acute mania a nephritis is the antecedent factor of which mania may be but a symptom. In delirium tremens the habits of the individual, the likelihood of pre-existing disease, the exposure and exhaustion attending a debauch leave little room for the older classification. In the case of the so-called reflex or ascending congestion, save in the rarest instances, we are prob-

Faulty classification.

ably dealing with an extension of infection. *General Causes.*—Sexual excesses, the excretion of large quantities of bile, and extreme concentration of the urine seem adequate to produce mild congestion. Fatigue, mental or physical, particularly when combined with exposure to cold or wet, would seem to be the most active exciting causes. Chief cause.

Morbid Anatomy.—The complex description and the varying terminology used by different writers and investigators lead to much confusion on the part of the student when he approaches the subject of renal pathology. The really essential factor should be clearly held in mind. All renal lesions, acute or chronic, fall under three heads:—(1). *Those in which glomerular changes predominate.* (2). *Those in which the interstitial changes are most prominent.* (3). *Those in which all structures are affected without predominance of any one group.* It must always be borne in mind that the finer classification and sub-divisions are of pathologic interest only, and that in any inflammation of the kidney, acute or chronic, one is likely to find all structures involved, so that it becomes simply a question of relative predominance. In an acute nephritis or severe acute congestion of the kidney, either glomerular, tubular or interstitial changes may be primary or predominant. With the exception of acute non-suppurative interstitial nephritis, the three fundamental structures are involved, and in any event, no clinical distinction can be made. We ordinarily find cloudy swelling, desquamation, and hyaline, dropsical and fatty degeneration or complete necrosis of the epithelium of the tubules, which are crowded with inflammatory detritus. We are likely to find acute intra-capillary glomerulitis, the capillaries being filled with cells and thrombi, and the epithelium of the tuft and capsule being involved. The latter is crowded with blood cells and leucocytes, and it will be readily seen that interference with renal circulation and nutrition, and hence with the urinary volume is inevitable, and further that the loss of integrity on the part of the filtration apparatus must necessarily lead to the escape of albumen. In rare instances a portion only of the kidney may be involved, or the process may be unilateral. The gross appearance of the kidney varies somewhat: it is ordinarily swollen and red, dripping blood on section, having a succulent feel, with an easily stripped capsule. More rarely it may be pale and mottled, exuding on section a milky fluid (acute non-suppurative interstitial nephritis). The glomeruli are usually red and prominent, but may be pale and indistinct.

Essential factors.

Clinical aspect.

Microscopic findings.

Macroscopic findings.

Symptoms.—The symptoms vary in severity with the type of the disease. An acute congestion may give few outward signs, and even an

Variable.

Pulse tension.

acute inflammation may first announce itself by uræmic convulsions or coma. One of the best guides in the case of nephritis complicating other diseases is found in the pulse tension, which is almost invariably increased in the presence of either acute renal congestion or inflammation.

Variable onset.

General Symptoms.—The onset may be sudden and frank, with chill and sharp temperature rise, or it may be gradual and insidious. There is usually some pain or heaviness in the loins and increased frequency of micturition, the pulse tension increases, and nausea, vomiting, headache and thirst may be present. The severity of the initial symptoms bears no definite relation to the type of the disease, save that those of simple acute congestion are ordinarily milder and more evanescent.

Early edema.

In some instances, the first thing noted is puffiness under the eye, the development of general edema, or even uræmic convulsions or coma. As a rule, however, these last symptoms occur only after several days of illness. *Fever* is ordinarily present, but is seldom high, and is often masked by that of a primary disease. In yellow fever the renal symptoms are often most striking and prominent. The *edema* of acute nephritis is oftentimes extreme, is likely to appear first in the eye-lids and tissues of the face and the skin is white and "pasty." In the extremities the swelling is comparatively firm, though pitting deeply on pressure, and because of the blue veining on the dead white background, is often termed "marble-like" edema. There is a special tendency to secondary complicating symptoms, especially affecting the serous membrane and the myocardium, and to attacks of edema of the glottis which may cause sudden death by suffocation.

Complications.

Special Precautions.—At least once daily the attending physician should by careful examination exclude endocarditis, myocarditis, pericarditis and pleurisy, with or without effusion, the condition last named often coming on rapidly and insidiously. Edema of the glottis usually demands immediate recourse to the most radical measures, emergency tracheotomy being the one usually found necessary. A *few moments* of indecision and delay has more than once caused death.

Glottic edema.

Urinary Findings.—It should be understood that the urinary findings in acute congestion vary with its severity but tend to assume, *pari passu*, identical characteristics. Severe acute congestion can be differentiated from acute nephritis only by its shorter duration, but the mildness of the slighter grades makes their recognition easy. The student should refresh his memory regarding the subject of albuminuria and the findings and significance of casts (pp. 324, 344) before taking up the following section. The findings fall naturally under three heads:

First Stage.—(5 to 10 days.) *Quantity for 24 hours* 100–400 c.c. *Color*:—dark, smoky or black. *Specific Gravity.* High or low, albumin may raise it to 1030; invariably low if albumin is removed. *Reaction.* Acid, or from large quantities of blood, slightly alkaline. *Solids.* Absolutely diminished, urea greatly diminished, chlorides may be absent if dropsy be extreme. *Sediment.* Abundant dark sediment. *Casts* abundant, dark granular, epithelial, fibrinous and blood casts with a few hyaline and fine granular. Casts carrying leucocytes, caudate pelvic cells, and round cells from calices. *Albumin.* ¼–1% according to degree of tubal involvement. Red blood cells abundant, leucocytes, brown granular epithelial cells.

Second Stage.—(5 to 10 days or more.) *Amount* 600–1500 c.c. *Color*, dark, smoky. *Specific Gravity* 1015–1020. *Reaction.* Acid. *Solids.* Still diminished. *Albumin* ⅛–¼%, diminishing pari passu with urine increase. *Sediment.* Profuse and dark; fatty changes now evident as fatty casts and renal cells and compound granule cells. The amount of fat indicates improvement and measures the severity of primary attack.

Third Stage.—Convalescence. Edema if present has now disappeared. *Quantity* 1500–4000 c.c. Tendency to polyuria, may last weeks. *Color.* Pale or slightly smoky. *Reaction.* Acid. *Specific Gravity.* 1006–1020. *Solids.* Normal or slightly increased by absorption of exudate. *Albumin.* Trace to ⅛ of 1%. *Sediment.* Scant, light color, abnormal blood cells (rings) (ghosts), occasional hyaline and granular (dark and light) casts, possibly an epithelial fatty or fibrinous cast. A few renal cells. Casts and renal cells may carry fat droplets or abnormal blood cells. As improvement continues the evidence of acute and regenerative changes disappears and urine is at last normal. Any chronic renal disease may show acute or subacute exacerbations.

Prognosis.—Less common and less fatal in dry than in damp climates. Children give large mortality, 30%, especially scarlatina cases. Low tension, uræmia, serous effusion are bad. Failure to improve after 10 days means liability to chronic disease. Persistent profound anæmia is a threatening sign. Recovery may take place even after one or two years. Relapse and multiple relapses frequent.

CHRONIC PARENCHYMATOUS NEPHRITIS.—Etiology.—An antecedent acute nephritis, prolonged exposure to wet and cold under conditions of fatigue, chronic malarial infection, syphilis, alcoholism, chronic suppuration are the common factors. **Morbid Anatomy:**—*Glomerular*, tubular and interstitial changes are present, the first pre-

dominating. The kidney is large, its capsule may be adherent, but usually strips easily from the pale and mottled kidney surface; section shows increased resistance and a swollen white cortex with curious areas of opacity. *Microscopic Findings.* There are obliterative hyaline degenerative changes in the vessels of the glomeruli, involving both the cells and the vessel walls and the tufts show swelling and nuclear proliferation. There is a tendency to connective tissue ingrowth and proliferation and desquamation of the capsular epithelium. The tubules show degeneration, desquamation and necrosis of their epithelium and edema and connective tissue infiltration of the intertubal tissues.

Symptoms.—The general symptoms are usually frank, pronounced anæmia and edema being prominent; the various uræmic phenomena are common and recurrent, there being a special tendency to headache, dyspepsia, intractable neuralgias, nausea, vomiting and diarrhœa. Pleurisy and pericarditis are common, ulceration of the colon occasionally occurs, and there is a constant tendency to increase of edema and to general anasarca. *The circulatory changes* are pronounced, the pulse tension is high, the second aortic and apical first sound markedly accentuated and the left ventricle hypertrophied.

Usually pronounced.

Urinary Findings.—It is customary to separate the urinary symptoms into those of the active, as compared with the inactive stage. *It will be noted that the essential symptoms are the same under both conditions*, i.e., a *decided albuminuria*, the presence of *fatty casts*, and marked *diminution of the urinary solids*. The disease is especially liable to subacute exacerbations, giving to the urinary findings a resemblance to an acute nephritis of the early stage of convalescence.

Frank.

Caution.

Active Stage.—*Amount.* 200–800 c.c. *Color.* High or pale, yellow or greenish, often smoky from exacerbation. *Reaction.* Acid. *Specific Gravity.* High from albumin, 1026–1035, but actually low. *Solids.* Greatly diminished, especially chlorides and urea as in all dropsical cases of renal disease. *Albumin.* Tremendous, tube may boil solid. ½–2% (5% has been reported, average 1%). *Sediment.* Always considerable, hyaline, granular, fatty casts, fatty epithelium and compound granule cells. Epithelial casts indicate superadded or recent or subacute trouble, free fatty cells, fatty crystals, cholesterin in late stages, ditto waxy casts, often red blood cells. **Inactive Stage.**—*Quantity* 800–1200. *Color.* Pale or greenish. *Reaction.* Acid or neutral. *Specific Gravity.* 1010–1015. *Solids.* Greatly diminished. *Albumin.* Large. ¼–½. *Sediment.* Practically as in active stage.

Prognosis.—If the disease is strongly intrenched and not merely a protracted case of acute Bright's disease, its victims rarely recover, but die in a few years of pulmonary edema, general anasarca or uræmia, the average duration of the disease being a little over two years.

THE SMALL WHITE KIDNEY.—This rare condition is supposed to represent an advanced stage of an unusually prolonged chronic parenchymatous nephritis with ultimate predominance of the interstitial elements.

Morbid Anatomy.—The kidney is small and yellow, because of the immense predominance of fatty change, connective tissue hyperplasia is marked, the capsule being thick and adherent, and the kidney surface under it rough and granular; on section there is increased resistance; the cortex is thin, pale, yellow, and covered with yellowish, white spots. *Microscopic Findings.* The interstitial changes are marked, there is arterial thickening, and the glomerular and tubule structures are degenerated and largely destroyed.

General Symptoms.—These are essentially the same as in parenchymatous nephritis, changes in the heart and blood vessels being more pronounced. **Urinary Findings.**—*Quantity.* Normal or slightly increased. *Color.* Pale. *Reaction.* Acid or neutral. *Specific Gravity.* 1004–1010. *Total Solids.* Greatly diminished. *Albumin.* ¼ of 1% or less. *Sediment.* Same as in chronic parenchymatous nephritis, save that casts are less abundant, and that waxy casts are found in unusual numbers.

CHRONIC INTERSTITIAL NEPHRITIS.—("Gouty" kidney, "contracted" kidney, "cirrhotic," "granular," "sclerotic" or "small red" kidney, "chronic diffuse" nephritis, etc.) Synonyms.

Morbid Anatomy.—The kidneys are usually small, an instance having been reported in which their combined weight was 1½ oz. The capsule is thickened and adheres to the dark red, nodular, granular surface, and section shows an increase in resistance. The arteries are prominent, the cortex very thin, the pyramids wasted and the pelvic fat increased. *Microscopical Changes.* These are essentially those of connective tissue overgrowth, with atrophy and degeneration of both glomerular and tubular structures. Many of the glomeruli and tubules are entirely destroyed. It is hardly worth while to distinguish an arterio-sclerotic kidney from a true interstitial nephritis. In the former, the change is primarily arterial, and the connective tissue changes are less diffuse, but in both, degeneration, atrophy, and destruction of the secreting structure is the essential feature. Arterio-sclerotic form.

Etiology.—Here, as in arterio-sclerosis and aneurism, the worship of Venus, Bacchus and Vulcan, form the primary factors in causation, though to this group may be added Mars, Minerva and Mammon. In other words, over-work, mental and physical, syphilis, sexual excess, exposure and privation, heavy eating and drinking, all play a prominent part. Arterio-sclerosis is a prominent feature in cases of interstitial nephritis, and one is not surprised to find what seems like a direct hereditary influence in the development of the latter disease. Lead poisoning, chronic malaria, gout and lithæmia, are common and prominent causative factors. As in the case of arterio-sclerosis proper, we find examples of the old young man, old by virtue of inherited vascular weakness or gout, or through sexual excesses, syphilis, or excessive mental strain.

Heredity.

General Symptomatology.—The pallor, edema and the frankly albuminous and cast filled urine of chronic parenchymatous nephritis makes mistaken diagnosis unpardonable, but the reverse is true in chronic interstitial nephritis. In the earlier stages of the disease a patient may appear to enjoy unusually good health, this being particularly true of the gouty type of the disease. Pallor, edema, and pigmentation are late symptoms, and the urinary findings are sometimes both variable and obscure. Many cases die unrecognized. In many more, the diagnosis is made only at autopsy and the various manifestations of uræmia may never be traced to their true cause. *Much dependence must be placed upon the secondary signs in the heart and blood vessels*, and the urinary examination demands care and the intelligent application of a full knowledge of the vagaries of this extraordinary disease. The general symptoms are essentially those of uræmia, which may be present at any time in any of its various forms, or may be postponed to the very end of the case. (The section on uræmia should be carefully reviewed.)

Insidious.

Slow.

Often unrecognized.

Uræmia.

Circulatory Signs.—*Advanced interstitial nephritis is invariably attended with increased pulse tension, as indicated by an over-acting or hypertrophied left ventricle, a hard radial pulse, and a marked accentuation of the aortic second or mitral first sound, with or without reduplication. Edema.* When marked edema appears in this disease, it is usually due to cardiac failure, and shows the characteristics of a passive congestion edema, the most dependent portion being first involved, as is the case in valvular disease of the heart. In many cases of interstitial nephritis, however, one may see the curious fullness of the eye-lids, especially the lower, presenting in the morning a tense appearance,

Important.

Essentially cardiac.

Suggestive sign.

the skin being thin, pearly, and almost translucent, while later in the day, the fluid may have disappeared and the delicate integument falls in fine wrinkles. Many of the cases fall under the head of a mixed nephritis, to be described more fully later on, and in these the renal facies is likely to be more marked and unmistakable, owing to the presence of a parenchymatous element.

Mixed form.

Changes in the Fundus.—In these as in other forms of renal disease, the eye changes may be pronounced and important, indeed, many cases are referred by the oculist under a correct diagnosis, without urinary examination. The usual changes consist of flame-shaped hemorrhages, papillitis, retinal edema, or peculiar fawn colored patches, radiating from the macula lutea. (See Fig. 1, Plate VII.) Glaucoma is not uncommon, and uræmic amaurosis may occur. As a rule, these eye changes indicate a fatal termination in a short period, yet the author has observed one case in which the original diagnosis was made by the oculist, and a subsequent glaucoma led to enucleation of the affected eye; yet the patient is still living after seven years in apparent good health.

Readily recognized.

Prognostic value.

Respiratory Tract.—It is important to remember the special liability of renal cases to attacks of bronchitis, pleurisy, asthma, edema of the glottis and dyspnœa in its various forms. A pleurisy with large effusion may come on so quietly as to attract no attention save through embarrassment in respiration. In a case recently observed the acute edema of the laryngeal tissues that followed a sharp attack of tonsillitis, proved to be due to an acute congestion superimposed upon an old nephritis of the arterio-sclerotic type associated with passive congestion.

Pleurisy, asthma glottic edema, etc.

Urinary Findings.—The essential symptoms consist in the increased amount of night urine, attended by increased frequency of micturition, a total increase for the 24 hours, a tendency to diminution in the total amount of solids, traces of albumin and a few hyaline and granular casts. Increased night frequency may be due to causes other than nephritis, such as enlarged prostate, chronic irritability of the neck of the bladder, or cystitis, but when the symptom is associated with a marked disturbance of the normal ratio between night and day, the symptom becomes one of primary importance.

Increased quantity.

Night vs. Day.

Diminution of Solids.—A man with interstitial nephritis may for years pass, the greater part of the time, a normal amount of urinary solids. In the latter stage of the disease it will almost invariably fall below normal, and this is especially true of urea. Variable information is often given by sudden drops in urea excretion and in a case under close

Urea variations.

observation, it is often possible to avert an impending attack of uræmia through this warning. *Albumin.* Albumin may be present continuously or intermittently, at one time of the day and not at another, but in uncomplicated cases will be found only in small traces. The specimens least likely to show it are those passed in the early morning, those most likely being the ones voided several hours after a full meal, taken in the middle of the day or a heavy dinner at night. Both physical exertion and the process of digestion seem to increase the albumin. *An examination of the single specimen, even of the 24 hours urine, proves no man free from interstitial nephritis. Color.* Usually pale. *Specific Gravity.* Low. 1002–1014. *Total amount* 2000–4000 c.c. May reach 7000 or 8000 c.c. *Reaction.* Faintly acid, or neutral. *Urinary Solids.* Diminished, coloring matter diminished, except indoxyl, which is usually increased. *Albumin.* Usually a trace, rarely reaches $\frac{1}{4}$ of 1% in the later stages.

Important data.

Typical cases.

Sediment.—Usually slight. Casts are chiefly hyaline and faintly granular. A few renal cells may be found. Some cases are associated with cylindroid storms.

Casts and cells.

Symptoms of the Terminal Stage.—There may be passive congestion due to a failing heart, resulting in an increased amount of albumin, and as a rule more numerous casts showing coarser granules and an occasional waxy cast. The amount of urine is diminished, the total solids are low, though the specific gravity may be relatively high. The general appearance of the patient is that of a cardiac edema, but it is extremely difficult to do anything for such a case.

Cardiac symptoms.

Urinary findings.

Prognosis.—No one knows for how long a period the changes of interstitial nephritis may endure in the living organism. It is safe to assume that cases have lasted for 40 or 50 years, and that the average duration is long. Through inheritance of cases, the author has been able to follow patients through a known period of at least 25 years, and is prepared to believe that some of them will add a decade more before the end comes. Cases are greatly jeopardized by the occurrence of subacute attacks which may be sufficiently severe to produce symptoms of acute congestion, or so mild as to show little more than a few blood cells or epithelial casts. The occurrence of the change in the back-ground of the eye, symptoms of cardiac incompensation and the appearance of waxy casts usually mean that the terminal stage has been reached.

Duration indefinite.

Subacute attacks.

Prognostic factors.

CHRONIC DIFFUSE NEPHRITIS WITH EXUDATION.—This disease represents clinically and pathologically a combination of

parenchymatous and interstitial forms. Many such cases are met with in practice. A common type.

Etiology.—The causes are those given for the individual lesion.

Symptoms.—*The general symptoms are those of chronic interstitial nephritis plus a more decided primary and terminal edema, a more varied sediment, and a marked anæmia.*

Urinary Findings.—*Quantity.* Usually increased, varying from 1500–3000 c.c. *Color.* Pale or with a slight greenish cast. *Reaction.* Neutral or acid. *Specific Gravity.* From 1004–1015. *Total Solids.* Markedly diminished. *Albumin.* Average between ¼ and ½ of 1%. *Sediment.* Hyaline and granular casts more abundant than in ordinary interstitial nephritis, and carrying fat in most instances, occasional fatty casts are found, and in advanced stages waxy casts. It will be noted that the symptoms throughout are those of parenchymatous and interstitial nephritis blended, and that they correspond accurately to the pathological changes. Mixed type.

AMYLOID KIDNEY.—*From the urine alone the diagnosis cannot be made* in this disease but the presence of conditions with which it is known to be associated, viz.:—amyloid degeneration of other organs, usually occurring in the presence of chronic suppuration or cachexias, is suggestive. *Polyuria* with a large amount of albumin, *hyaline casts* in variable numbers, associated not infrequently with granular and occasionally fatty and waxy casts, constitute the urinary findings, the most significant feature of which is the usual combination of *polyuria, low specific gravity and a large albumin content.* Basis of diagnosis.

MOVABLE AND FLOATING KIDNEY.—The normal kidney may or may not be palpable, but usually its lower border may be detected under proper conditions of muscular relaxation and correct technique. To palpate the kidney, one hand should be placed over the floating ribs behind, the other below the costal margin on the outer side of the rectus in the mammillary line. By steady firm pressure the two hands should be approximated, and during full inspiration allowed to separate slightly when the movable kidney may be felt to pass between them, and may be directly engaged and palpated during the manœuvre. No attempt should be made to grasp the kidney primarily, and in any event, it should be engaged between the fingers of the two hands by simple approximation rather than by any grasping or clutching movement such as is sometimes recommended. Though, ordinarily, such an examination can be carried out when the patient is in a dorsal position, it is often useful to examine them in a position Technique of palpation. Caution. Patient's attitude.

Author's preference.

between the dorsal and the lateral, the arm on the side under observation being allowed to hang loosely forward, and the patient receiving some support to relieve the abdominal tension.

Degrees of Renal Displacement.—The palpable kidney is one whose lower edge can just be felt by the examiner. Such a kidney is not abnormal; the movable kidney is that which slips back and forth like a "pea in a pod," or one which can be fixed by passing the examining fingers above its superior border during full inspiration. The term floating kidney is applied to those having more than a vertical displacement, or such as are vertically displaceable to a lower level than the umbilicus. The range of mobility is often extraordinary and in some instances the kidney may be found in the pelvis.

Movable vs. "Floating."

Etiology.—The disease may be congenital, but is ordinarily acquired. It is far more common in women than in men and most often observed in multipara. About three-fourths of the cases occur on the right side and in about one-seventh the condition is double. Congenital relaxation of the ligaments, tight lacing and repeated pregnancies, the wasting of the peritoneal fat of the capsule, traumatism, muscular strain and other alleged causes must be placed in the "not proven" class. It is probable that in many cases a combination of these causes may be operative. A curious relationship seems to exist between movable kidney and appendicitis, the two conditions being often coincident.

Dubious factors.

Symptoms.—Strange to say cases of extreme movability may exist without any symptoms, while slight cases may cause the most intense crises and no adequate explanation of this fact has ever been given. It is often wise to withhold information from the patient where no symptoms seem to be present, or where the condition is incidentally or casually encountered. When symptoms are present they vary from those of mild neurasthenia and dyspepsia to the remarkable crises first described by Dietl. *These are attacks sudden in onset, characterized by severe abdominal pain, nausea, vomiting and in extreme cases by chill, fever, and even symptoms of collapse.* The utmost care should be observed in diagnosis, as errors are astonishingly frequent. In one case coming under the author's observation, a young unmarried woman had double floating kidney. She was subject to attacks of violent pain referred to the region of the gall bladder, and associated with jaundice. A noted surgeon was so far misled as to operate for cholelithiasis without result. In another case, precisely similar attacks occurred without jaundice, and the case was referred to a surgeon for a fixation operation. Symptoms continued after operation and a sub-

Curious variability.

Dietl's crises.

Misleading cases.

sequent incision revealed a gall bladder full of stones. In another instance a co-existing appendicitis was found to be the real source of the pain. The author ventures to express the opinion, founded upon somewhat extensive opportunity for observation, that *true Dietl's crises are relatively rare; and that the appendix, gall bladder or gastric ulcer are more likely to be essential factors.* Cases have been reported in which the condition has been confounded with renal colic, but such an error should seldom occur. The co-existence of profound neurasthenia and hysteria often complicate the examination. The association of movable or floating kidney with the displacement of other abdominal viscera is described under Glénard's disease.

Dietl's crises rare.

Frequent associations.

PYELITIS AND PYELONEPHRITIS.—*Definition.* By a pyelitis is meant an inflammation completely or chiefly confined to the pelvis of the kidney, by pyelonephritis an inflammation involving both the kidney substance and the pelvis. The former can hardly exist without a slight invasion of the kidney texture. **Etiology.**—Various micro-organisms are capable of causing these conditions; amongst these are:—the pyogenic streptococci, staphylococci, typhoid and colon bacilli, gonococci and tubercle bacilli. Infection in these cases is usually ascending, the bladder or ureter being the primary source. They may, however, arise in connection with certain virulent acute infections, or in those of the chronic sort, and under these conditions, the infective agent would appear to be brought direct by the circulation. Renal calculus is a potent cause, yet stones may exist for years in a kidney without causing any marked disturbance.

Usually ascending infection.

Symptoms of Pyelitis.—An acute pyelitis is ushered in by fever, pain in the back, or tenderness in the region of the twelfth rib, and usually marked by frequent micturition. The urine in acute cases is diminished in amount, of high acidity, and contains pus in quantity. The pain even in simple suppurative pyelitis may be so severe as to simulate renal colic (see page 63), and may radiate in the same way to the groin, inner side of the thigh or testicle. Chill and fever may occur at any time during the course of an acute process; certain cases have been mistaken for malarial fever on this account. Occasionally, the ureter of an affected kidney becomes blocked, and retention on the part of the unsound kidney permits the excretion of normal urine from the sound side. So, also, masses of stringy pus and debris may produce obstruction at the neck of the bladder, and pain simulating that of stone in the bladder. The urinary sediment shows large quantities of pus, a variable amount of blood, usually of the slightest

Septic and renal.

Colic.

Temporary blocking.

Sediment.

quantity and the more or less characteristic cells of the renal pelvis (see figs. 135, 137). Ordinarily one will find also a few renal cells and an occasional cast. Under prompt and efficient treatment these symptoms rapidly subside so that in a period varying from a few days to two or three weeks, an uncomplicated case may recover.

Symptoms of Pyelonephritis.—These are essentially the same as those of pyelitis, save that the amount of pus is usually greater and sediment shows a decidedly greater number of renal elements. It will be noted that the characteristic features consist of (a). *An acid urine.* (b). *Containing pus.* (c). *Yielding a sediment showing characteristic elements*, i.e., casts, renal and pelvic epithelium. The picture may be much obscured by the presence of a cystitis in which case the urine may be ammoniacal, and the sediment may be so profuse, and with such predominance of the cystic elements as to greatly complicate the diagnosis.

RENAL TUBERCULOSIS.—This occurs in two forms, the *acute miliary* which is merely a part of a general tuberculosis and the so-called *caseous form* which is of great clinical interest. The latter commences usually as a miliary or larger nodule which undergoes much the same changes as would occur in other portions of the body. Either one or both kidneys may be affected and the process frequently involves the whole urinary tract including the urogenital apparatus.

Forms.

Extent.

Frank vs. Latent.

Diagnosis.—The diagnosis may be extremely easy or surprisingly obscure, the development being in some cases that of a *frank tuberculous pyelitis or pylonephritis*, in others pursuing a latent course with no symptoms save those of irritation. *In every case attention should be given to the organs so often secondarily affected, such as the spermatic cord, testis, prostate, and in the female the ovaries and tubes.* So also persistent dysuria without signs of bladder disturbances sufficient to account for the condition will oftentimes prove to be of tuberculosis origin. In most cases there is a slight elevation of temperature and in a goodly number tuberculosis, present or past, or sufficient family history is made evident .Symptoms of lithiasis may co-exist and complete the picture. The affected kidney may or may not be movable primarily and in some instances becomes greatly enlarged and readily palpable. The author's experience has not been such as to permit him to coincide with the opinions expressed by some of our best surgeons that the tuberculous change is of necessity a progressive and destructive one. In a considerable number of instances of slight involvement and several which presented a double tuberculosis so

Dysuria.

Temperature.

Associated lesions.

Not always progressive.

advanced as to forbid operation the process has shown the same tendency to arrest as is so often observed in the lungs. *Finally, the diagnosis must depend upon the finding of the tubercle bacilli in the urine and their differential staining by proper methods and it must be remembered that they may be found in urines showing but the slightest traces of pus.** In some instances the use of tuberculin is justifiable and may be followed by an appearance of the bacilli in the urine. Tuberculin test.

Renal Tumors.—Their general characteristics have already been described on page 231 and the subject is one for surgical rather than extended medical discussion.

HYDRONEPHROSIS.—*Definition.* By hydronephrosis is meant an over-accumulation of urine, within the kidney and some portion of its ureter, due to obstruction. A persistence of this condition may result in a conversion of a kidney into a large cyst. **Etiology.**—The condition may be temporary or permanent, persistent or intermittent, and it may be either congenital or due to disease. Amongst the causes are stricture of the ureter or urethra, calculi, clots, a twisted ureter, such as occurs in floating kidney; or from neoplasm in the genito-urinary tract, or involving structures in close relation to it. The condition is commonly temporary and intermittent, but due in most instances to temporary torsion, pressure or removable obstruction.

ACUTE CYSTITIS.—Etiology.—An acute inflammation of the mucous membrane of the bladder may be due to injury or irritation from calculi, foreign bodies, wounds and the introduction of sounds, or may be associated with urethritis, tuberculosis, prostatitis or mere exposure to cold and wet, sexual excess or the toxæmia of infectious diseases. **Symptoms.**—Dysuria with increased frequency and marked tenesmus are the chief symptoms, though pain may be severe and radiate to the perineum, the glans, hypogastrium or thighs; fever is usually slight and may be absent. *The urine* is scant, strongly acid, of variable, usually high specific gravity and contains an approximately normal amount of solids, albumin, blood and pus in variable quantity. The sediment consists of pus and blood cells in quantity with much bladder epithelium and numerous round cells.

CHRONIC CYSTITIS.—This may result from an acute attack or may insidiously develop as the result of enlarged prostate, stricture, the frequent use of the catheter, infection from the genitals (female) and of course from calculi, growths or tuberculous process affecting the viscus.

* In two cases recently observed the urine was almost clear and the only subjective symptom a slight dragging pain over the kidney.

Like every other hollow, muscular organ, the changes may be either atonic or hypertrophic and in many cases of long standing the bladder is greatly contracted and tends to become incrusted with urinary salts. **Symptoms.**—These are those of the acute form, usually in a milder degree both as to subjective symptoms and urinary findings, though in the latter blood is less likely to be present in quantity, and the reaction is more likely to be alkaline and show the presence of the volatile agent, ammonia. The solids are usually somewhat diminished, the sediment will show triple phosphate and often ammonium urate crystals in the presence of an alkaline reaction.

Comment.—It should be noted that these cases lack all evidence of renal involvement and that the albuminuria present is usually in direct proportion to the blood or pus from which it is derived.

TUBERCULOSIS OF THE BLADDER.—A chronic cystitis presenting no symptoms of stone, stricture or enlarged prostate should always suggest tuberculosis. It usually involves primarily the trigone and the urethral orifices. The seminal vesicles and prostate should always be examined for nodular infiltrations and weight should be given to recent hemorrhages. **Symptoms.**—The appearance is that of a severe or mild chronic cystitis, with or without hemorrhage, but associated usually with acid urine. The diagnosis depends upon the finding of the tubercle bacilli, and the use of the cystoscope.

TUMORS OF THE BLADDER.—These fall properly under surgery and it need only be said that papillomata are the most frequent, aside from malignant growths of advanced age, and that they produce chiefly symptoms of chronic cystitis associated with marked intermittent hæmaturia. Occasionally bits of the growths may appear in the sediment, together with shreds of tissue and caudate cells from the villi of the growth.

ACUTE PROSTATITIS.—This may result from the same causes that lead to an acute cystitis and is recognized by the swelling, heat and tenderness of the gland, associated with throbbing, pain in the back and legs, dysuria and constant urgency, both rectal and vesical, with marked tenesmus and increase of pain attending the end of urination. Casts of the prostatic ducts may be present in the sediment and occasionally spermatozoa, otherwise the urinary picture resembles acute cystitis. **Complications.**—Prostatic abscess may develop and is usually associated with known symptoms of sepsis and may cause mechanical retention of urine.

CHRONIC PROSTATITIS.—This is most frequent at advanced ages

in connection with chronically enlarged prostate but may also follow acute attacks or be associated with chronic posterior urethritis. The association of modified symptoms of the acute form associated with palpable hypertrophy or swelling of the prostate and the symptoms of chronic cystitis make the diagnosis.

Chronic Urethritis.—Need not be considered in this volume.

URINARY CALCULUS.—This most commonly occurs in the bladder or renal pelvis but stones may occupy the ureters and vary in number from one to several hundred, and in size from a mere grain to that of a large orange. They vary greatly in form, usually assuming the shape of the cavity in which they lie and occasionally being polished by attrition when several lie in contact. Uric acid, cystin and phosphate present usually a smooth surface and the calcium oxalate stone is lobulate and rough (mulberry calculus). Uric acid and urates vary in color from pale yellow to deep brown, phosphatic stones are grayish or white, those of calcium oxalate deep brown, of cystin, yellow. Section should always be made to determine the constituents as several concentric layers may be found. As to frequency, uric acid and urate stones predominate. In all forms the urinary sediment is likely to furnish suggestive findings during the stage of stone formation containing the characteristic crystals of uric acid, ammonium or sodium urate or calcium oxalate or triple phosphate crystals, etc., according to the nature of the process.

Tests for Urinary Calculi.—The concentric layers should be sawed through and tests made from the scrapings of the different layers and of the powdered nucleus. The following table is taken from Dr. J. B. Ogden's admirable book.*

CHEMIC EXAMINATION OF URINARY CALCULI.

1. **Preliminary Examination.**—Heat on platinum foil:

Albumin=a flame with odor of burnt horn.

Urostealith=a flame with odor of shellac and benzoin.

Cystin=a blue flame with odor if SO_2.

Xanthin and uric acid=char without a flame.

Alkaline urates=alkaline residue soluble in H_2O.

Earthy phosphates=a residue soluble in acetic acid *without* effervescence.

Calcium oxalate and calcium carbonate=a residue soluble in acetic acid *with* effervescence.

* Clinical Examinations of the Urine and Urinary Diagnosis.

Calcium carbonate=original powder soluble in acetic acid *with* effervescence.

Calcium oxalate=original powder insoluble in acetic acid.

Silica=residue insoluble in HCl.

Murexide Test for Uric Acid.—Original powder + HNO_3 and evaporate = pink residue + NH_4OH = purple color=uric acids and urates.

Original powder + HNO_3 and evaporate + KOH=violet color, which disappears on heating=uric acid. Violet increases on heating=xanthin.

2. **Systematic Examination.**—Presence of uric acid shown by (1). Boil in H_2O and filter.

A. Filtrate + HCl. Let stand 24°=crystals of uric acid. Bases in solution. Concentrate.

Calcium urate=one drop of solution + solution ammonium oxalate=crystals calcium oxalate.

Magnesium urate=one drop of solution + NH_4OH + Na_2HPO_4 =crystals ammonio-magnesium phosphate.

Sodium urate=one drop of solution + $Pt.Cl_4$=after concentrating, prisms of sodioplatinic chloride.

Potassium urate and *ammonium urate*=one drop of solution + $Pt.Cl_4$=dodecahedra of potassioplatinic chloride and ammonioplatinic chloride.

Potassium Urate.—Evaporate solution and ignite on mica. Residue + HCl + $Pt.Cl_4$=potassioplatinic chloride

Ammonium Urate.—Evaporate solution and ignite on mica. Residue=no crystals with $Pt.Cl_4$.

B. Portion insoluble in H_2O. Add HCl.

Uric acid=insoluble.

Calcium carbonate=soluble with effervescence. Filter + NH_4OH=precipitate of calcium oxalate, calcium phosphate, and ammonio-magnesium phosphate. Wash. *Calcium oxalate*=insoluble in acetic acid. Filter + ammonium oxalate to filtrate. *Calcium phosphate* gives precipitate of calcium oxalate. Filter + NH_4OH to filtrate=precipitate of ammonio-magnesium phosphate.

GLYCOSURIA.—(Sugar in the Urine). *Clinical Definition.* In a clinical sense glycosuria comprises all conditions under which the urine contains grape-sugar (glucose) in sufficient quantity to respond to the

ordinary tests. In short, *all diabetics have glycosuria, but all who have glycosuria are not diabetics.* Sugars of various kinds may be found in the urine, but glucose is the one of especial interest to the physician. Normal urine contains it, as it does albumin, but not to an extent that can produce any confusion in clinical work; *hence any urine that shows sugar when tested, according to methods here recommended, is abnormal.* Furthermore, glucose or dextrose may appear either persistently or intermittently in the urine of apparently healthy persons and no sharp line separates the benign from the pathologic condition, save that suggested by Stengel, who says that all cases fall under one of two classes, viz.:—1st those easily controlled (simple glycosuria), 2nd those that are intractable (diabetes mellitus). The urine of one who takes into a fasting stomach phloridzin or large quantities of glucose, a woman recently delivered, or the man with a brain injury may temporarily contain sugar. The stout high living individual may carry such a condition for many years (lipogenic diabetes) without serious impairment of health; but *lapse of time alone suffices to prove the benign nature of any given case.* Glycosuria may entirely disappear for long periods, only to recur, and not infrequently to assume a malignant orm. Such periods of latency cannot be justly estimated, and the very causes that are assigned for benign glycosuria are quite commonly associated with the development of diabetes mellitus. The conditions under which diabetes develops are in brief the following:—**Age.**—It occurs at all ages, but chiefly in the fifth and sixth decades. Its prognosis is inversely as the age of the individual. Children rarely recover, and in them, the course may be astonishingly acute. **Sex.**—80% of the cases occur in males. **Race.**—Certain races suffer greatly from disease. In Tunis and in Malta its ravages are comparable to those of tuberculosis in European countries. This, however, applies chiefly to city dwellers. Hebrews are especially liable to the disease. An enormous increase has been noted in certain cities during the last three decades. In Danish cities and in Paris, for example, the mortality is said to have quadrupled during that period. Due allowance must, of course, be made for improved diagnostic methods. **Heredity** is very marked. Schmitz reports that diabetes has occurred in the blood relations of 998 of the 2115 individuals whose cases he had investigated, fourteen cases having been reported in one family. **Diet.**—Digestive glycosuria is under certain conditions quite unimportant. Van Noorden would place a definite limit to the amount of sugar that should be disposed of under normal conditions, without producing glycosuria, and various

Glucose.

Intermittent cases.

Important.

Temporary recession.

Etiology.

Dietetic.

writers have suggested a test amount varying from 100 to 250 gm., this to be taken at one sitting, and on a fasting stomach. If sugar then appears in the urine, the individual is a suspect. It is said that in such cases the sugar excreted is of the same variety as that ingested. **Exercise.**—There can be no doubt that a rich diet, associated with sedentary habits and great mental activity, are potent factors in causation.

Arthritic glycosuria.

Gout.—Gout is distinctly associated with diabetes, though it is claimed that the arthritic form is comparatively benign. The reason for such an association is evident, the same habits of life and hereditary elements being present in both diseases. It should be noted, however, that the consumption of alcohol is not often a prominent factor in the causation of glycosuria. **Diseases Antecedent or Complicating.**—Aside from those mentioned in the opening paragraph, gout, tuberculosis and nephritis are commonly associated with diabetes, the two latter being usually secondary conditions. **Obesity.**—A large number of glycosurics are obese, and this lipogenic form is ordinarily easily controllable and often curable, nevertheless, many cases apparently benign at the outset become true diabetes. **Pancreatic Disease and Sclerosis of the Spinal Cord.**—It is probable that about one-third of the cases of diabetes are pancreatic in origin. Sclerosis of the cord may be secondary or primary, and the relationship of the two conditions has not been sufficiently worked out.

Lipogenic.

The three P's.

Symptoms.—The so-called "three P's," Polyuria, Polyphagia, Polydypsia, represent the fundamental symptoms as seen in a typical case and the thirst and increased frequency of urination and large amount of urine are usually the first symptoms noticed. An excessive appetite is seen in nearly all advanced cases. Rapid loss of weight and strength is marked in true diabetes and often prominent in the benign forms. *The Skin.* The skin is dry, eczema, boils, carbuncles are common and gangrene is often a terminal complication. *The Eye.* Neuro-retinitis, hemorrhage and cataract are often seen. *Muscles.* Besides pronounced weakness, muscle cramps are common in advanced cases. *Sexual Organs.* Impotence, amenorrhœa, pruritus vulvæ and balanitis occur and pregnant women are likely to abort. *Nervous System.* Neuritis, headache, mental irritability or depression, drowsiness or insomnia may occur, true melancholia may develop, and in 50% of the severe cases the knee jerks are absent. *Lungs.* Pulmonary tuberculosis is a common terminal event.

Coma.—Diabetic coma may be the first recognized symptom of the disease. The author recalls a casual meeting on the street which led

to the recognition of unsuspected advanced diabetes in the infant child of a friend, the babe dying a few hours later in diabetic coma. In this case the breath had the peculiar aromatic sweetness present in all cases of threatening coma and presumed to be due to the presence of acetone. Other premonitory symptoms are headache, drowsiness, nausea, vomiting, dyspnœa and rapid pulse, the dyspnœa is a definite air hunger, the term accurately describing the symptom. Constipation is often marked and the symptom of mild or severe indigestion. In actual coma the breathing may be slow and the deep sighing, superficial or Cheyne-Stokes. Certain cases are characterized by profound collapse, and in others ataxic symptoms are prominent. *The Blood.* Bremer's test may be valuable in coma if for any reason a specimen of urine is unattainable. Two smears, one of diabetic blood, the other of normal blood are made in the ordinary manner. They are fixed by heat and stained for two minutes in a 1% aqueous solution of congo-red. The diabetic smear remains unstained or shows a pale or greenish yellow. The control smear of normal blood is stained red. If methylene blue is used the diabetic smear takes a yellowish green, the normal blue. On the other hand, a solution of Biebrich-scarlet stains the diabetic smear scarlet and does not affect the normal blood. These blood reactions may reveal a glycæmia in the absence of glycosuria.

Important signs.

Bremer's test.

Glycæmia.

The Urine.—Total quantity increased, the amount sometimes reaching or exceeding 20,000 c.c. The specific gravity is high, varying from 1030 to 1060. Sugar varies from a mere trace to 8 or 12% in extreme cases.

Quantity and weight.

Important Variations.—It must be remembered that in glycosuria as in albuminuria the abnormal constituent of the urine may be totally absent for considerable periods, may be lacking at one time of the day and present at another, and that it may be wholly removed by careful dieting and proper medication, thus making it possible for a glycosuric to pass a life insurance examination. Furthermore, *the specific gravity is not always high*, and a specific gravity of 1010 does not justify the omission of the test for sugar, as is so generally believed. No one of the cardinal symptoms is absolute, polyuria is not an invariable symptom and as might be inferred both polyphagia and polydypsia may be absent and even in true diabetes long periods of latency may occur. The tests for sugar are dealt with on page 333.

Removable sugar.

Low specific gravities, etc.

Diacetic Acid and Acetone.—See page 338.

Prognosis.—The prognosis in glycosuria depends primarily upon the decision as to whether one is dealing with true diabetes or simple

glycosuria. If the former, the prognosis is always grave, if the latter, is favorable. In youth, the disease is almost invariably rapidly fatal, cures being extremely rare. The thin offer a more serious prognosis than the fat. The presence of diacetic acid is a dangerous signal calling for prompt measures of elimination such as would be used in threatening uræmia. Apoplexy, carbuncles, tuberculosis, gangrene and coma are distinctly terminal events.

Important factors.

DIABETES INSIPIDUS.—This curious ailment, occurring usually during the first three decades of life, and more frequently in the male, may be induced by traumatism, violent emotions, acute disease, tuberculous peritonitis, sunstroke, cerebral syphilis or tumor, diseases of the abdominal viscera, including aneurism. Heredity is sometimes evident and it may be congenital. **Morbid Anatomy.**—The lesions consist merely of congestion and dilatation of the kidney and ureters, and hypertrophy of the bladder. Various lesions of the nervous system have been reported but none specific. **Symptoms.**—Excessive thirst, striking emaciation and a dry skin co-exist with the passage of enormous quantities of pale, faintly acid or neutral urine (5000 to 20,000 c.c.) during the 24 hours. The specific gravity is extremely low (1001 to 1005), yet the total solids for the 24 hours are greatly increased, urea often exceeding 100 gms. Albumin and casts if present at all are scant and of no significance and the sediment is negative ordinarily.

Etiology.

Great quantity of light urine.

Solids increased.

Differential Diagnosis.—The disease is most commonly confused with interstitial nephritis but the mistake can seldom occur if the total solids are estimated as should invariably be done, an increase being the rare exception in the latter disease. Furthermore, the cardio-vascular symptoms of nephritis in any case likely to be confounded with diabetes insipidus would be marked.

Prognosis.—This depends upon the cause of the disease. If associated with cerebral tumor or organic disease in any part of the body it is bad. Many of the idiopathic cases recover.

DISEASES DEPENDENT UPON OR ASSOCIATED WITH CHANGES IN THE BLOOD AND DUCTLESS GLANDS.

Examination of the Blood.—**Hæmatology.**—This recently developed branch of medicine has proven invaluable to the internalist and useful to the surgeon, affording *a means of exact diagnosis in malaria, the leukæmias, filariasis, relapsing fever, typhoid fever, Asiatic cholera, Malta fever* and *trypanosomiasis, fundamental evidence in cases of pernicious anæmia, chlorosis, secondary anæmia, Hodgkin's disease and*

Scope and value.

diabetes mellitus and valuable corroborative evidence in the acute pneumonias, appendicitis, septic infections, trichiniasis, lead poisoning, gout, malingering and certain cases of malignant disease. Indeed these are but a part of the large group and in addition one must consider the *valuable information represented by negative findings.* Thorough laboratory training and accurate, painstaking work are essential to success and superficial or theoretic knowledge is of little value.

Clinical Essentials.—(a). *The determination of hæmoglobin.* (b). *The red cell count.* (c). *The white cell count.* (d). *The examination of fresh blood.* (e). *The examination of the smear preparation* (a. *stained,* b. *unstained*). (f). *The study of the various "clump" or agglutination reactions in certain acute infections* (serum diagnosis).

Clinical Tests of less Importance.—(a). Counting blood plates. (b). Estimation of alkalinity. (c). Cryoscopy. (d). Specific gravity determination. (e). Coagulation period. (f). Determination of blood volume and ratio of corpuscles to plasma.*

Terms in Common Use.—*Anæmia.* A deficiency in corpuscles, coloring matter, or total blood volume. *Oligocythæmia.* A deficiency of red cells. *Oligochromæmia* A deficiency in hæmoglobin. *Oligæmia.* Deficient blood volume. *Leucocytosis.* The term is commonly used to denote an abnormal increase in the number of leucocytes the polymorphonuclear type predominating. The average per cubic millimeter is normally about 7,500. *Lymphocytosis.* An unusual increase in the number of mononuclear leucocytes. *Leucopænia.* The opposite of leucocytosis and equivalent to *hypoleucocytosis. Plethora.* A term covering what was formerly supposed to be a pathological condition, but now used to indicate an increase in the total quantity of blood. The term " plethoric" is applied to those individuals with a ruddy countenance due to dilated or unusually prominent capillary network. The condition is now of slight clinical importance, though it is certain that there is a direct relation of the blood volume, to the musculature and the size of the heart. *Polycythæmia,* an abnormal increase in the number of red cells may, rarely, be a symptom of importance, particularly when associated with enlarged spleen and cyanosis (Osler's disease) or acetanilide addiction.

* This section naturally reflects not only the author's personal experience but the teaching of Thayer, Simon, Cabot, Hewes, DaCosta, Ewing and E. S. Wood as conveyed by their published work, or, in the case of the first four, by personal instruction or communications. Nevertheless the publications of Ehrlich, Grawitz, Sahli, T. Laache, Neusser, Hayem, Durham, Scott and others have been freely consulted.

Term defined.

Color Index.—The color index represents *the result obtained by dividing the hæmoglobin percentage by the percentage of red cells*, the normal for hæmoglobin being 100; and 5,000,000, cells in men, or 4,500,000 in women, being the assumed normal for erythrocytes.*

To Obtain Blood for Examination.—It is essential that the actual technique should be thoroughly understood, and intelligently and expeditiously carried out.

Sterilization.

Precautions.—One should not trust to the flow of blood for sterilization. The ear lobe or the finger tip should be carefully cleansed with alcohol, bay rum, cologne, or some similar substance always available, and *the needle or lancet used should be sterilized in the flame. The patient should be asked whether he bleeds easily* or the reverse with a view of determining the size, depth of the puncture, and of avoiding

Bleeders.

serious complications in cases of hemophilia. Two cases of this kind in the author's clinic bled steadily for several hours from a very minute puncture. For making the puncture an instrument with a sharp cutting

Best instrument.

edge should be used. The Hagadorn needle and the spring lancet *with a trocar point*,† are most suitable, the ordinary needles being poor substitutes. The puncture should be sufficiently free and deep to obviate *squeezing or excessive friction*, and furthermore, the instru-

Method.

ment should be used with a sharp, quick painless stroke,‡ and the first two or three drops should be discarded. The blood should flow spontaneously and to maintain it *light* friction may be used, *but pressure never*, and to check it firm steady pressure is sufficient, or in the case of the finger, vertical elevation of the arm.

Care and Preparation of Slides and Cover-glasses.—All material

Form and size.

should be of the best sort, $\frac{7}{8}$ square cover-glasses being superior to the round, and both these and the slides should be *thin*. When received from the dealer, they should be thoroughly washed in soap and water and then placed in the following solution:—Hydrochloric acid, 1 part, absolute alcohol, 29 parts, water 70 parts, or they may be placed in

Preparation.

solution containing equal parts of alcohol and ether.|| When required,

* *Example* 1. In a given case the hæmoglobin is 30%, the red cells number 3,000,000, or 60%. 30 divided by 60 equals $\frac{5}{10}$, a low color index indicating chlorosis. *Example* 2. Hæmoglobin 30%, red cells 1,000,000 or 20%. 30 divided by 20 equals 1.5; a high color index such as is found in pernicious anæmia. *Example* 3. Hæmoglobin 50%, red cells 2,500,000 or 50%. 50 divided by 50 equals 1; color index indicates secondary anæmia.

† To be obtained of any instrument house.

‡ Blood may be taken from young children in this way without arousing them from sleep.

|| The rapid evaporation of the latter constitutes an objection to its use.

they should be wiped thoroughly, polished with tissue paper or with soft linen, and, if convenient, passed through a flame. A small drop of blood will spread satisfactorily and evenly between a perfectly clean slide and its cover-glass or between two cover-slips, especially if these are slightly warm.

Use.

Fig. 146.—Blood smear. Ordinary method.

Making the Smear.—To make a good smear preparation the cover-glass must be absolutely clean and a small drop only should be lightly touched by one, the other placed quickly upon it, an instant allowed for the spread of the blood and the two separated by a sliding (not a lifting) movement as shown in figure 146. The cover-glass should never touch the skin. Another method consists in using the edge of one cover-glass and drawing it lightly across the drop, another of a similar procedure with microscope slides and in the use of the cigarette paper edge as shown in fig. 148. *A good smear should show the cells evenly distributed throughout large areas and should not look to the eye smeary or thick.* If carefully preserved *the dry unstained specimen will keep for a long time*,* a matter often lost sight of by the physician. Forceps are not needed at any stage of the procedure but the stained preparations are more brilliant if the slip is immediately dried over a flame.

Correct technique essential.

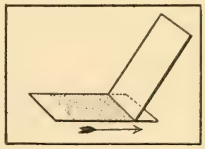


Fig. 147.—Making blood smear with two cover-slips.

Fixation of Dried Preparation.—The almost universal use of the Wright stain makes a prolonged description of the older methods unnecessary. For rapid work with the triple stain it is sufficient merely to pass the smear through the flame rapidly 30 or 40 times with the clean surface down, much as in fixing the preparation for staining tubercle bacilli but with rather less heat. The same results may be obtained by boiling specimens for a minute in absolute alcohol, by fixing by one minute's immersion in 1% alcoholic solution of formaline, or by a few seconds' exposure to formaline vapor. The older heating methods are delicate, require for good results a special oven and are not necessary for ordinary clinical work.

Rapid fixation and staining.

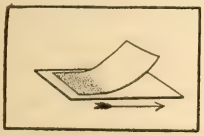


Fig. 148.—Cigarette paper method of preparing blood films.

Examination of the Fresh Blood.—Fresh blood is readily examined

* Specimens in the author's possession have stained well after one or even two years, but there is a time limit, recent efforts to stain some six year old smears proving futile.

by allowing a small drop to spread evenly under a perfectly clean cover-slip and slide, both of which have been gently warmed. It may afford information of the greatest value in regard to the following conditions:—(a). *Leukæmia.* (b). *Malarial organisms* (and their amœboid movements if the stage is kept warm).* (c). *Filariasis sanguinis hominis.* (d). *Relapsing fever.* (e). *Trypanosomiasis.* (f). *Rouleaux formation.* (g). *Increase of fibrin.* (h). *Increase of blood plaques.* (i). *Low color index.* (j). *Approximate number of red and white cells.* (k). *Cell deformity.* Nucleated red cells are not to be distinguished by this method, and while it is *possible* for an expert to make a *rough* differential count of unstained leucocytes, such a step is seldom necessary or advisable. The presence of "blood dust" or hæmochronia is indicated by a rapid vibration of these tiny bodies, which in all probability correspond to extruded cell granules. By sealing the edges of the cover-slip with a little vaseline one may preserve the fresh specimen for hours.

Extruded granules.

Examination of the Stained Specimen.—The selective affinity shown by the different constituents of the cellular elements of the blood for certain aniline dyes affords a ready means of cell differentiation. The aniline dyes are divided into 3 groups (a) *basic*, (b) *acid*, (c) *neutral*. By using dyes of the various classes in combination in a single stain one may in a single step attain differentiation. The basic dyes, such as methyl violet, methylene blue or hæmatoxylin (chromatin stains) act chiefly upon nuclei. Acid dyes, such as eosin, orange G. or acid fuchsin are protoplasm stains, while neutral dyes (resulting from the mixture of acid and basic colors in solution) color beautifully the "neutrophile" granules of the leucocytes. All modern methods of staining depend upon the recognition of this selective affinity.

Basis of staining methods.

Staining Solutions.—Up to within a short time the favorite blood stain was Ehrlich's triacid or triple stain, but this has now been displaced by Wright's modification of Jenner's stain, an alkaline eosinate of methylene blue, which not only produces beautiful results, but at once *fixes* and *stains* the specimen, thus eliminating the laborious and delicate heating process of the older method.†

Wright's Modification of Louis Jenner's Stain.—The student or practitioner had best purchase a solution ready made through some

* Brownian movement and the contractions preceding crenation and cell death can hardly be confounded with true amœboid movement.

† The old eosin and hæmatoxylin or eosin and methylene blue mixtures are quite out of date in clinical work.

drug supply house, as the formula is somewhat complex,* and it should be kept tightly corked to prevent precipitation.

Technique of Staining.—(1). The dried but unfixed smear, completely covered with the stain is left one minute. (2). Then distilled water is added to the stain on the cover-slip drop by drop *until a greenish metallic scum appears and the margins show a reddish tint.* (3). After *three minutes* the stain is washed off with water, leaving a purplish specimen, which is *washed until the film is yellowish or pink,* (4) gently dried between filter paper, and (5) mounted in balsam.

Rapid and simple.

Results.—The appearance of the various cellular element is as follows:—*Erythrocytes.* Orange or pink throughout. *Erythroblasts* show deep blue nuclei, *blood-plaques* purple, *mast cell granules* deep purple, *polymorphonuclear leucocytes* show lilac or blue nuclei, *neutrophile granules* lilac, *eosinophile granules* pink, fine *basophile granules* a deep blue stain, *bacteria* and such organisms as malarial parasites blue.

Action on cells.

Ehrlich's Triacid Stain.—The ready mixed *powder* for making this stain may be bought of any supply drug house and the student or practitioner should not attempt to make the original:—Formula: Ehrlich-Biondi powder gr. xv. Alcohol (*absolute*) 1. c.c. Distilled water 6 c.c. Fairly good rapid work may be done with a good triple stain in the absence of Wright's stain, by heating the smear carefully as in the staining of tubercle bacilli and staining for 30 sec., washing, drying and mounting (Cabot). Other heat fixation methods are now so little used that a description is not deemed necessary. Scott fixes by a few seconds' exposure to formaline fumes before applying Jenner's stain and shows beautiful results.

Hæmoglobin.—Hæmoglobin or more properly oxyhæmoglobin constitutes about 9-10 of the bulk of the red corpuscles. It is a proteid substance containing 4% of hæmochromogen, an iron holding body, and 96% of concentrated, almost insoluble albumin *which readily*

* *Formula.*—(a). Make a 0.5% aqueous solution of sodium bicarbonate, place in Erlenmeyer plaque, add 1% of Grübler's medicinal methylene blue; place in steam sterilizer for an hour. (b). After cooling, add while stirring with glass rod, a 1–1000 aqueous solution of Grübler's water-soluble yellowish eosin until the color of the original mixture changes to purple and presents a lustrous yellowish scum upon its surface. (About $\frac{1}{2}$ as much eosin solution as methylene blue solution will be found necessary.) (c). Collect this scum by filtration, dry it, and with it saturate methyl alcohol. (100 c.c. of the latter will dissolve about $\frac{3}{10}$ of a gram of the dry precipitate). (d). Filter and add 25% of methyl alcohol. The stain is now ready for use *and should neither precipitate nor show impairment though kept for a long period.*

forms unstable compounds with oxygen. The great value of its determination by clinical tests may be readily appreciated.

Tests for Hæmoglobin.—*An expert observer can make a close estimate of the percentage of hæmoglobin from the appearance of the ordinary stained or even the unstained smear preparation* as may be readily appre-

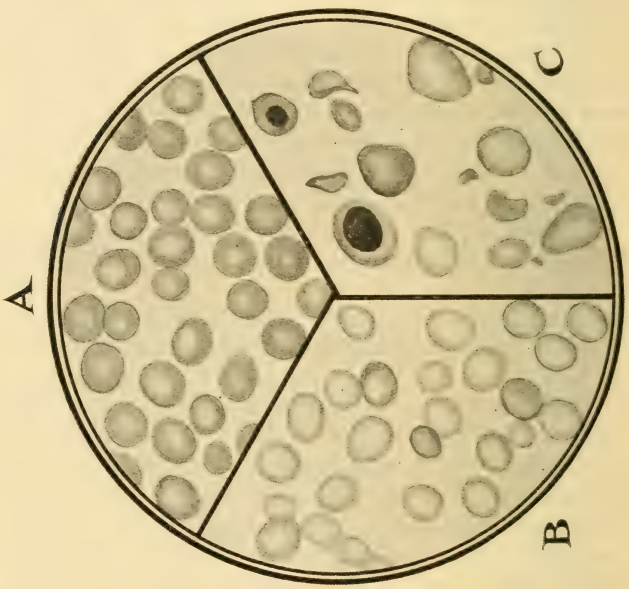


Fig. 149.—A. Normal blood. B. Chlorosis. C. Pernicious anæmia. The plate shows the sharp contrast between cells normally rich in hæmoglobin and the light cell of chlorosis and also the poikilocytosis and marked variation in size noted in pernicious anæmia. (A normoblast and megaloblast also appear.) Stained smears.

ciated by referring to fig. 149, but for accurate work several forms of apparatus have been devised, *all depending upon a comparison of a given specimen of blood, either whole or in a known degree of dilution, with a fixed color scale.*

Tallqvist's Hæmoglobinometer.—This, the simplest of apparatus, consists of lithographed color bands, each with a central perforation, and represents the color of blood in dilutions running from 10% to normal, when applied to absorbent paper.* A drop of the patient's

* The color scale is bound with 50 sheets of special paper each divisible into three parts, furnishing material for 150 tests.

blood is taken up by the absorbent paper and the resulting stain is placed under the central perforation of the color bands and comparison is made *as soon as the stain has lost its wet gloss*, not after complete drying. This is a simple, rapid but inaccurate method allowing probably 10% variation, and the color scale fades, if not kept from the light. It is distinctly inferior to Dare's instrument which takes hardly a minute more of time.

Dare's Hæmoglobinometer.—A circular disc of tinted glass, representing variations in blood coloring matter of a known degree, is brought into direct contrast by transmitted candle light with a film

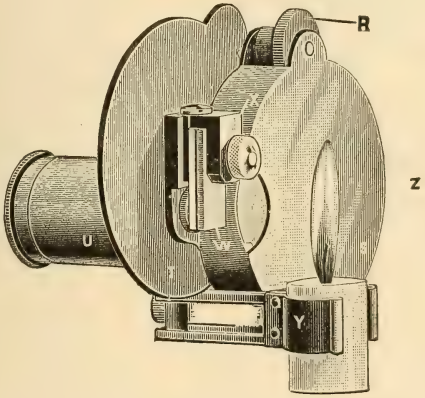


Fig. 150.—Dare's hæmoglobinometer. U. Observation tube. T. Shield. W. Removable plate with capillary opening for holding blood. X. Thumb screw for holding same. Y. Candle holder and candle. R. Milled wheel for revolving color disc. V. Aperture for illumination and color comparison. S. Case inclosing color disc.

of the fresh whole blood drawn by capillary attraction between two glass plates, one transparent, the other translucent and white. A detachable observation tube and a circular shield, protect the eyes from extraneous light, and the percentage of hæmoglobin may be read directly from the scale. The instrument may be used in daylight, but should be pointed at some dark object and its readings are not affected by an excess of leucocytes.

Von Fleischl's Hæmoglobinometer.—This well known instrument or its more accurate modification has been largely superceded by the

simpler and cheaper instrument of Dare. If it is used one should procure Miescher's modification. The following precautions are necessary to good results:—(a). *The capillary tubes* for taking the drop *must be absolutely clean* and should be tested out for equality of calibration when purchased. (b). *All blood must be removed from the surface* before mixing, *leaving the calibre exactly filled.* (c). *The blood must be taken quickly, washed out into the chamber, and thoroughly mixed in the shortest possible time.* (d). *The observer should face the end of the movable*

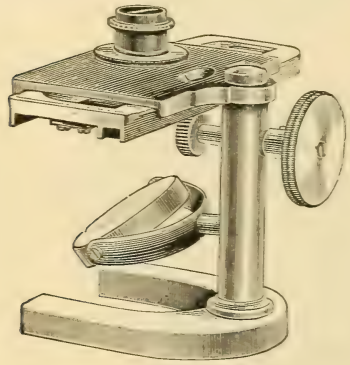


Fig. 151.—Von Fleischl's hæmoglobinometer. Description. Milled wheel at right moves a tinted glass wedge under the fixed metal stage surmounted by double chamber reservoir which receives light from the calcium sulphate reflecting disc below. Half of the same chamber is filled with distilled water into which is stirred the blood contained in the measuring capillary pipette. The other contains only plain water but receives its light from the colored wedge. By moving the wedge back and forth the colors are matched and the percentage reading is shown on a scale visible through the opening just in front of the supporting upright.

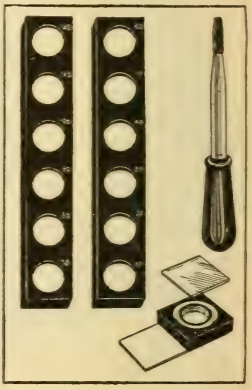


Fig. 152.—Oliver's hæmoglobinometer. The discs shown as white in the illustration are colored to represent the various blood dilutions and direct comparison is made with a solution of the actual blood obtained by a measured mixing capillary pipette furnished with the instrument. Intermediate readings are obtained by placing squares of tinted glass over the fluid under examination.

colored wedge with the thumb screw on his right. (e). *Decision as to color should be made quickly* to avoid uncertainty and confusion. (f). *The thumb screw should be sharply turned in order to obtain as vivid a contrast as possible* until the final match of color is achieved. (g). *Should the blood solution appear turbid and lack proper color, as in the case of leukæmia, add a few drops of a dilute aqueous solution of potassium hydrate.* (h). *Where the hæmoglobin is below 30%, double or treble the usual amount of blood should be used,* the percentage obtained being

divided proportionately. (i). *The examination should be made in a dark room, or by means of a light proof box and in any case some form of tube should be used for observation,* a simple roll of black paper being ordinarily sufficient.

Hæmoglobin Estimation by Specific Gravity.—By the use of tables to be found in all the larger works dealing with this subject the hæmoglobin present in a given specimen may be very accurately estimated by Hammerschlag's modification of Roy's method. The procedure is too cumbersome and fussy to be recommended to the practitioner and will not be described. Unnecessary.

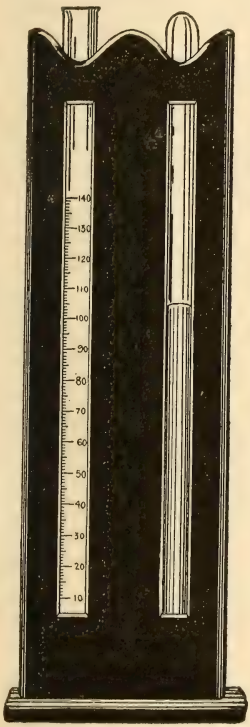


Fig. 153.—Sahli's hæmoglobinometer.

Oliver's Hæmoglobinometer.—As shown by fig. 152 this instrument depends upon the scale of colors based upon diluted blood. Its use is sufficiently indicated by the diagram and it has no advantage over the two preceding methods.

Gower's Hæmoglobinometer.—This little instrument has the merit of extreme simplicity, yet in practice one may lose more time by its use than with the other more elaborate instruments, inasmuch as a slight error in dilution means a repetition of the whole process.

Sahli's Hæmoglobinometer.—This admirable instrument resembles Gower's hæmometer but is the best of all forms for accurate work. The empty *graduated* tube is filled to the mark 10 with a decinormal solution of HCl which is saturated with chloroform. An excellent instrument.

The blood is then added by means of a measuring pipette and the mixture made complete. Finally distilled water is added until the color corresponds to that of the control tube.

The color comparison is remarkably easy and definite.

The instrument should be kept in its case or in a dark place.

THE ERYTHROCYTES AND THE LEUCOCYTES.

The Red Blood Cell.—Each cubic millimeter of human blood should

Normal.

contain from 4,500,000 to 5,000,000 erythrocytes, the former number representing the average for the female, the latter that of the male. A fluctuation of half a million cells may be assumed as within normal limits. The average size of these cells is 7.5μ but a wide variation is possible (6.5μ to 8.5μ). Under pathological conditions one may meet with *extremely large or extremely small cells*, and with *nucleated erythrocytes* (*erythroblasts*).

Abnormal.

Classification of Leucocytes.—The simplest modern classification deals with six varieties:—(1). *The polymorphonuclear neutrophiles* which constitute from 60–75%. (2). *The small lymphocytes* (20–30%). (3). *The large lymphocytes*, and (4) the *transitional forms* (4–8%). (5). *Eosinophiles* (0.5–5%). (6). *Basophiles* (0.5%).

Six varieties.

(1). The *polymorphonuclear neutrophile* cell measures 7.5–12μ and is characterized by its irregular, dark blue staining nuclei, often connected by chromatin strands, and the presence of neutrophilic granules, taking a lilac or pink color with Wright's stain. (2). *The small lymphocyte.* This contains a single round deep staining nucleus almost filling the cell and often obscuring the border of protoplasm. It measures 5–10μ. (3). *The large lymphocyte.* This measures 10–15μ and carries a round or oval, pale blue staining nucleus and a relatively large amount of protoplasm. It differs from the transitional leucocyte only in its indented crescentic or reniform nucleus. (5). *The eosinophiles* (8–11μ) are readily recognized by their coarse, rose colored granules and polymorphonuclear type. (6). *Basophiles.* (7.5–12μ.) The distinguishing feature of the basophile cell is its fine deep blue granules. The nucleus is polymorphous and stains a dull blue. The characteristic granules are best shown by Wright's stain and not at all by the triple stain.

How recognized.

Unusual or Abnormal Forms.—*The myelocytes.* These leucocytes may contain either neutrophilic or eosinophilic granules constituting two sub-varieties. The nucleus is spherical or oval, tends to be eccentric, stains feebly with the blue, occasionally shows division and often shows a marginal light circle. *The presence of the granules distinguishes it from the large mononuclear forms and the lack of lobulation or twisting of the nucleus from the polymorphonuclear cell.* With Wright's stain the neutrophilic myelocytes show lilac or pink granules with a purplish admixture. Eosinophilic myelocytes show the ordinary eosinophilic granules. *Significance.* The cell is invariably pathologic, occurring in many cases of profound anæmia or excessive leucocytosis, occasionally in lymphatic leukæmia and Hodgkin's disease, but only in small numbers,

Nucleus and granules.

Disease association.

PLATE II.

Chief varieties of cells encountered in health and disease (Wright's stain). 1. Normal red cell. 2. Common form of polymorphonuclear leucocyte. 3. Lesser lymphocyte. 4. Eosinophilic myelocyte. 5. Eosinophilic leucocyte. 6–6. Neutrophilic leucocytes: upper left, transitional form, on right neutrophilic myelocytes. 7–7. Large lymphocytes. 8. Normoblast. 8. Normoblast showing division of nucleus. 9. Normoblast nucleus. 10–11. Basophilic leucocytes. 12. Megaloblast.

Giant "blast."

of fine structure which stains a faint blue or green and in triple stain the specimen is surrounded by a white definitive ring. The nucleus is usually large, more rarely strikingly small and deeply staining like the normoblast or microblast. It represents defective hemogenesis and

Associated conditions.

signifies degeneration, being found as a *prevailing type of erythroblast only in primary pernicious anæmia, nitro-benzol poisoning*, and *bothriocephalus anæmia*. In any severe anæmia an occasional cell of this type may be found.

Microblast.—This cell (5–6μ) presents essentially the same appearance, aside from size, as the normoblast and has the same clinical significance.

Unusual Forms.—Various mixed types are encountered (mesoblasts) which defy differentiation, showing contradictions in size and staining reaction; some which DaCosta regards as immature normoblasts show a pale nucleus with acid stain stippling and polychromatophilic protoplasm. Yet others, large in size, contain a small deeply basic staining nucleus and faintly stained protoplasm. These he would classify as megaloblasts. Cabot has described and drawn extraordinary *intra and extra cellular ring bodies* in lead poisoning, pernicious anæmia and lymphatic leukæmia. They have also been found by DaCosta in the profound anæmias of sepsis. Basic ring bodies were previously described by Strauss and Rohnstein. It is now known that these for the most part take the acid stain of Wright's solution, rarely the blue.

Meso-blasts.

Ring bodies.

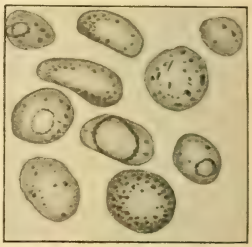


Fig. 154.—Basophilic stippling and Cabot's ring bodies. (After Cabot, Ewing and DaCosta.)

Basophilic Erythrocytes.—May be encountered as fine, coarse, spicular or even ovoid granules occurring in regular or irregular distribution and occurring in lead poisoning, chlorosis with intestinal auto-intoxication, practically all of the profound or pernicious forms of anæmia, but absent in the anæmia of renal and hepatic disease, the acute infections, syphilis and diabetes.

Significance.

The Blood Cell Count.—The differentiation with the various types of anæmia requires that the cells contained in 1 cm. of any given blood be directly and differentially counted and this is readily done with the modern apparatus. *The Thoma-Zeiss hæmocytometer* consists of two graduated capillary pipettes, i.e. the erythrocytometer and the

Differential value.

leucocytometer for counting red and white cells respectively. The blood is drawn by suction or capillary attraction from the drop obtained by puncture, until it reaches the point marked 1, then *rapidly* wiped dry and the diluent *quickly drawn in* until it fills the bulb and reaches the mark 101.* While drawing in the diluent the pipette is revolved between the finger and the thumb to set in motion the mixing bead contained in the bulb and when filled again thoroughly shaken and revolved for half a minute. This mixing should be repeated each time before expelling a drop for examination. One has then a mixture, each drop of which presents a blood dilution of 1:100. Many prefer a 1:200 dilution readily obtained by using the mark 0.5 as the upper limit for the indrawn blood. The blood rises quickly if the pipette is perfectly clean and may exceed the proper level if not watched or checked by withdrawing the point and quickly wiping it. If the higher dilution is used any excess is readily blown out or better drawn down by touching the tip with filter paper or blotter. The next step consists in expelling the diluent occupying the capillary portion of the tube, after which a small drop of the mixture in the bulb is placed upon the central disc (shown in fig. 155), the cover-glass is placed in position and the cells given time to settle. The drop should just or very nearly fill the central plateau without running over into the moat around it when the cover-glass is applied, else the process must be repeated after thoroughly cleaning and drying the cover-glass and counting surface. At the point of contact between the cover-glass and the underlying plate concentric color rings (*Newton's rings*) should appear indicating perfect contact. Their absence vitiates the count and indicates dust or moisture which must be removed and the process repeated. The specimen being placed under the microscope carrying a Leitz No. 6 objective and a No. 4 ocular or their approximate equivalents should show an even

Instruments and technique.

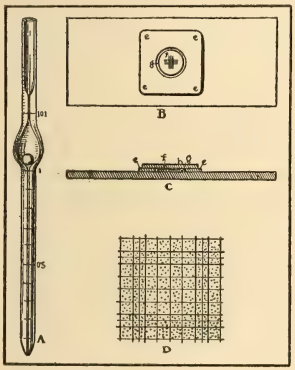


Fig. 155. — Thoma-Zeiss blood counter, showing pipette, counting chamber and ruled field.

Magnification.

* The diluent used is Toisson's solution. Formula:—Methyl violet 5 B (0.025), sodium chloride (1.0), sodium sulphate (8.0), neutral glycerine (30.0), distilled water (160.0). Hayem's solution contains mercuric chloride (0.25), sodium chloride (0.5), sodium sulphate (2.5), distilled water (100.0).

distribution of cells throughout *both the ruled and unruled fields*. There appears on examination a magnified ruled field with squares appearing as in fig. 155. Up to this point a certain amount of blood diluted 100 times has been placed upon a field so ruled that each of its squares represents an area of 1-20×1-20 mm. i.e. each side of the square represents 1-20 mm. A third dimension has been added by the cover-glass brought into contact with the blood but lying 1-10 of a mm. above the level of the depressed central disc which carries the squares upon its surface. One has now to count the number of cells lying within a definite number of the small squares, and for the sake of accuracy it is best to include the whole 400 though for ordinary work a less number may be taken if the blood seems to be perfectly distributed.* One should pursue a systematic course crossing from left to right and back from right to left and so on, counting not only the cells that lie within each square but those that touch its upper and left hand sides. Having counted the cells, say in 100 squares, one multiples by 4000 if the original dilution was 1:100 and by 8000 if it were 1:200, the result representing the number of cells in 1 cm. of the blood under examination.† The blood counter must in every case be cleaned *immediately after using*, (1st) wtith water, (2d) alcohol, (3d) with ether, and the glass bulb should be absolutely free and movable after the process is completed. If to be again used at once, air should be blown through the tube by means of a hand bulb and rubber tube, not the mouth. The counting chamber should be cleaned with cold water only and should not be exposed to direct sunlight for fear of softening the cement.

Basis of method.

Important precaution.

The Leucocyte Count.—For this the large calibred pipette is used with a diluent consisting of an aqueous solution of *glacial* acetic acid (0.5%) which leaves visible only the white cells. The most accurate counts are made by using 0.5 rather than 1 as the basis of the dilution, and even this requires a good sized drop. Furthermore, the pipette must be kept nearly horizontal at the time and after the diluent is added, else it will run out of the tube. The diluent is then added to the point 11 and the count can be made with the erythrocyte counting chamber or better with Zappert's chamber in which the central square (*one millimeter*) is

Instruments and technique.

Zappert's chamber.

* For the highest degrees of accuracy the *whole process* is repeated several times and an average taken, but this is ordinarily impossible in clinical work.

† Assuming that 1200 erythrocytes have been counted in 100 squares in 1 square there are 12 cells. This is multiplied by the dilution, i.e. 100 or 200, and by 4000 because the microscopic squares represent but $\frac{1}{4000}$ of the cubic mm. used as the standard.

surrounded by 8 unruled squares *of the same size.* The total obtained by counting the leucocytes in the 9 squares is multiplied by 200 and divided by 9, assuming a dilution of 1:20 as above described. If the *ordinary counting chamber* is used the process is greatly facilitated by inserting an eye piece diaphragm into the tube of the ocular which will cut off exactly 100 of the small squares. These may be bought or can be made by the physician from metal or cardboard by the physician. The leucocytes in 400 small squares of the erythrocyte chamber may then be rapidly counted and as many additional outside diaphragm fields as are necessary to correct work. Accurate white blood counts require patience and the counting of a large number of fields, for a slight error has a disastrous effect.* Usually two drops of the 1–10 or four drops of the 1–20 dilution will enable one to count 140–150 cells.

Basis of count.

Use of erythrocyte counter.

Drops required.

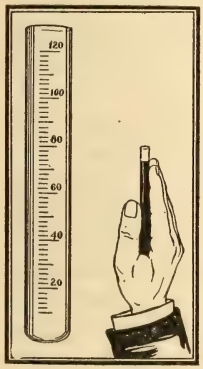


Fig. 156.—Method of holding Oliver's hæmatocytometer.

Oliver's Hæmocytometer.—This ingenious instrument is shown in fig. 156, and, save in cases where the white count is desirable or in leukæmia where the excess of leucocytes vitiates any optical method, is convenient and accurate. *Method.* A small amount of Hayem's solution is placed in the glass tube and into it is stirred the blood taken up by the capillary pipette. It is then diluted gradually with the Hayem's mixture until a bright horizontal line becomes visible as the observer looks through the mixture at a candle flame beyond. Each point on the scale represents 50,000 red cells.

Ingenious and convenient.

Technique.

Precautions.—(a). One looks at the edge of the tube, not its flat faces. (b). The pipette should be thoroughly clean before using. (c). The tube should be inverted to secure proper admixturc each time that the diluent is added. (d). An imperfect marginal line forecasts the appearance of the complete transverse line and warns one to go slowly with the dilution. (e). A small Christmas candle should be used in a dark room. (f). The tube should be close to the eye and the observer fully ten feet from the candle.†

The Hæmatocrit.—(Hedin-Daland). This consists of a graduated capillary tube, each degree of which represents 100,000 erythrocytes.

*The ingenious self-filling and self-measuring pipette of Durham has many advantages, but requires separate mixing vessels.

†Cabot finds the instrument highly inaccurate in *severe* anæmia and unavailable in *leukæmia* but otherwise it is an excellent instrument for erythrocyte determination.

Approximate results.

The tube is filled with blood and placed (with a similar tube opposite) in the centrifuge which should be revolved at high speed for at least two minutes when the red column will represent the erythrocytes present as indicated by the scale. The tube when filled by suction is dried and closed at its other end by the vaseline coated finger which is kept in place while the rubber suction tube is withdrawn, and results obtained are rapid but probably only approximate.

Laborious but valuable.

Differential Counting.—This involves the examination of from 500 to 800 leucocytes and a determination of the exact number of each variety present. The field should be systematically and carefully gone over, and if ordinary care is observed, the procedure offers no difficulty for one who is sufficiently skilled to recognize the various types of white cells and the abnormal erythrocytes which should be included in any such procedure. The process is a tedious one involving as a rule the counting of 2 or 3 entire stained smears.

LEUCOCYTOSIS AND LYMPHOCYTOSIS.—Assuming that the term leucocytosis covers an increase of the polymorphonuclear neutrophilic cells both relative and absolute, it is evident that *one must know first what constitutes a normal leucocyte count; second, what conditions other than disease may increase the number. The Normal Average.* This varies from 5–10,000 to the c.mm. of peripheral blood, the average being 7500. **Physiologic Leucocytosis.**—The most important is the

Digestive.

so-called digestive leucocytosis which represents on the average an increase over normal of about $\frac{1}{3}$. The variation is greater after a heavy proteid meal than on a vegetable diet and reaches its maximum usually within from 2 to 4 hours after a meal. The increase may be purely polymorphonuclear or more rarely is common to all normal forms.

Preagonal.

It may be delayed in cases of subacid dyspepsia and is very marked in nursing infants and diabetics. *The so-called preagonal leucocytosis* may be observed immediately before death, the counts occasionally reaching 20,000 or even 30,000 cells. Cabot states that in pernicious anæmia this may take the form of so decided a lymphocyte increase as to simulate lymphatic leukæmia, nevertheless as a rule it is a polymorphonuclear increase due probably to the stasis of a failing circulation.

Pregnancy.

It is important to remember that both the later months of pregnancy in primiparæ and the first week following delivery a moderate leucocytosis is the rule.

Newborn babes.

So also in newborn babes the count usually ranges from 50–20,000 during the first two days of life and that after the second week during the first year it remains at 10–15,000 per c.mm. The type of physiologic leucocytosis is almost invariably polymorphonuclear

(85 to 90%) though there may be a coincident lymphocyte increase in the newborn, malignant disease, or in hemorrhage.

The Leucocytosis of Disease.—So many diseases are associated with a more or less marked leucocytosis that it is only necessary to enumerate some of the more striking instances *and more useful to remember the exceptions* or more particularly the cases in which a diminution (hypo-leucocytosis) occurs. *Practically all inflammations*, particularly of serous membrane, and *any suppurative process attended by* toxæmia would ordinarily produce leucocytosis, yet we may have these, lacking such a phenomena because (a) *the inflammation or toxæmia may be too slight to excite a reactive change*, (b) *because the toxin overwhelms the organsim*, thus preventing leucocytic reaction, (c) *because the organism is primarily too feeble to respond.* In illustration one may cite lobar pneumonia as representing a disease in which leucocytosis is ordinarily marked, and typhoid fever in which a leukopænia is ordinarily found. These diseases stand at opposite poles, the one being sthenic and the other asthenic, the one of brief duration, the other prolonged and tedious, the one showing early and profound toxæmia, the other allowing its victim to escape by resolution before the system is overwhelmed by toxins. Acute appendicitis offers another illustration. In this disease one may have an inflammation and toxæmia so slight as to produce no leucocytosis, and overwhelming toxæmia with the same result, or, as is the rule, a well defined leucocytic reaction representing good vitality opposed to sharp infection. It is useless to attempt to name or even tabulate the conditions giving rise to leucocytosis. In abscess it is practically constant, but in *malignant disease* a high count may be found only in rapidly growing tumors of the kidney, lungs or liver, and in the author's experience especially in metastatic hepatic invasion from gastric carcinoma. In *ptomaine poisoning, uræmia, cholæmia*, and gas poisoning the leucocytosis may be marked. So also in cases of *acute delirium, convulsions, after surgical operations* or indeed *after general anæsthesia.* The blood findings of the various diseases are discussed under the individual headings.

General conclusions.

Examples.

Various associated conditions.

Leucopænia.—(Hypoleucocytosis). This covers *any leucocyte count running below* 5000 *cells*, excluding of course those cases of reduction associated with profound anæmia. As might be expected this condition may be present as a result of malnutrition or starvation, but its chief importance is its occurrence in certain diseases of the infectious and toxæmic type. Amongst these are *typhoid, paratyphoid, malaria, Malta fever, influenza, measles and rötheln, tuberculosis* (in the absence of sep-

Highly important.

tic symptoms), *leprosy*, *trypanosomiasis* and less commonly in *chlorosis*, *chronic infantile gastro-enteritis*, and, following the use of *agaricin*, *atropin*, *camphoric acid*, *ergot*, *picrotoxin*, *sulphonal and tannic acid*.

Lymphocytosis.—*This may be either relative or absolute*, the former without any necessary increase in the total white cell count, *the latter covering both an increase in percentage and in the total lymphocytes. The latter is practically always a lymphatic leukæmia.* It should be remembered in this connection that in infancy the lymphocytes and transitional cells may constitute 55 or 60% of the total white cells, gradually dropping to the normal figure at the age of 4 or 5 with a corresponding increase in the polymorphonuclear forms, hence the average increase percentage (50–70) has little significance at early ages. *Either the large, small or transitional forms may predominate in any case and the condition is invariably pathologic when occurring in older children or in adults*, and is usually associated with profound malnutrition, and appreciably marked in the terminal stages of diseases of that type as well as in marked anæmia, and in several of the acute and chronic infections characterized by hypoleucocytosis, notably *malaria, typhoid, Malta fever, scarlet fever, diphtheria, measles, tuberculosis and trypanosomiasis. So also we find the condition marked in ailments involving the lymphatic glands or spleen, or the invasion of lymph channels by malignant growths.* As regards drugs it is said to follow the administration of *thyroid, tuberculin, quinine and pilocarpin.* Its constancy in syphilis in which it is combined with eosinophilia is of diagnostic importance.

Normal of infancy.

Abnormal.

Syphilis.

Eosinophilia.—The determination of this condition is made by a differential count establishing their proportion associated with a count of the total leucocytes, the latter multipled by the former giving the total number of eosinophiles in a c.mm. *It is of lessened value as a clinical sign because present in a great number of diseases but its constancy in trichiniasis, hydatid disease and in the presence of the more important intestinal parasites is of some value.* The attempt made by some of the foreign hæmatologists to create a group of which eosinophilia should be diagnostic has not proven successful. *It is*, as stated, *of clinical value in syphilis when combined with lymphocytosis.*

Melanæmia.—Pigmented leucocytes occur in cases of melanotic sarcoma and in malaria, free in the blood only during the segmentation stage of the latter disease.

Iodophilia.—If a dry but unfixed smear be *gently* pressed down upon a generous drop of specially prepared iodine and potassic iodide solution*

* Iodine 1. Potassium Iodide 3. Water 100. Gum Arabic 50.

the normal blood cells take a uniform yellow tint but in *septic conditions* both intra and extra cellular brown granules are seen or a diffuse protoplasmic staining occurs, especially affecting the neutrophilic leucocytes and, rarely, the basophiles and myelocytes. Sepsis.

The reaction is *reasonably constant* in septic conditions *other than pure tuberculous abscesses*, but is present in many other diseases, such as purpura hæmorrhagica, acute miliary tuberculosis, certain typhoids and malignant disease. It is of genuine value in the differentiation of septic from non septic effusions and joint disease. Other diseases.

Perinuclear Basophilia.—Neusser's perinuclear basophilic granules occurring in specimens submitted to the triacid stain are by him regarded as indicating the uric acid diathesis but are generally regarded as accidental products of no diagnostic significance.

Agglutination Reaction.—Our present understanding of the phenomena constituting the so-called agglutination reaction which have proven so valuable in the diagnosis of certain obscure diseases depends upon the known fact that if erythrocytes or other cells not those of the animal into whose blood they have been introduced, or bacteria similarly injected, produce in the blood serum of the animal so treated the peculiar quality of agglutinating and precipitating, *in vitro*, the foreign elements. This involves the assumption of *agglutinins* and *precipitins* as products evolved during adaptation or immunization. The process has proven of value in a number of diseases and is best illustrated by typhoid fever in which disease is found its first clinical use through the work of Widal based upon the previous investigations of Pfeiffer, who worked along purely bacteriological lines seeking to apply the phenomena to the identification of bacteria. *The agglutination reaction of typhoid, paratyphoid, colon infections, dysentery, cholera, plague, Malta fever and to a much less degree of pneumonia and tuberculosis* have proven valuable. An early and definite reaction is chiefly marked in typhoid, Malta fever and cholera. In paratyphoid one is sometimes embarrassed because of the fact that the paratyphoid bacilli of different epidemics may not react. In dysentery there are three types of bacilli to be dealt with, viz.:—those of Flexner, Shiga and Hiss, while in plague the reaction is not constant and occurs too late to be of use, and the same objection of inconstancy applies to tuberculosis and pneumonia.*

Basis.

Value.

Diseases yielding reactions.

Confusing factors.

* The student is earnestly advised to read in this connection a description of Ehrlich's side-chain theory of immunity, an excellent description of which may be found in DaCosta's book, "Clinical Hematology, 1905," page 151.

Counting the Blood Plates.—This requires a diluting fluid* through a drop of which placed upon the finger the puncture is made. After mixing, the count is made by the Thoma-Zeiss counting chamber to determinate the ratio of plaques to erythrocytes. It is then only necessary to count the erythrocytes to know the number of plaques present in the given specimen.

Coagulation Time.—It is sometimes important to estimate the coagulation period in hæmophilia, purpura and in "obstruction of the biliary tract with or without jaundice" (Cabot) as in these conditions clot formation may be greatly retarded. Wright's capillary tubes may be used or the simple method represented by fig. 157. In the former coagulation should occur within from 3–6 minutes, by the simpler method from $2\frac{1}{2}$–5. The latter merely involves the placing of several drops of the suspected blood upon slightly warmed microscopic slides which are tilted upwards at varying intervals until they appear as in B of the plate, the elapsed time representing the coagulation period.

Fig. 157. Delayed coagulation of blood, a simple test. (After DaCosta.)

The Determination of Alkalinity.—Some personal experience with this method has convinced the author that it does not merit attention as a *clinical* procedure.

Miscellaneous Methods of Blood Examination.—Ingenious methods for the estimation of erythrocytic vulnerability, determination of the total blood volume, of fat and fatty acids, of osmotic tension, of iron viscosity, etc., are interesting but are not available, and, for the most part, not important for the general practitioner.

CYTODIAGNOSIS.—Widal has given this name to the study of the leucocyte content of pleural, peritoneal and cerebro-spinal fluids as they are obtained in operative or diagnostic aspiration. It is a valuable procedure within narrow limits. The fluid must be centrifugalized, and if immediate attention cannot be given it, any clot may be broken up by shaking in a flask with small glass beads or the fluid may be drawn into a syringe or aspirator flask half full of a solution consisting of calcium oxalate (2 parts) and normal saline solution (1000 parts. *Salhi*). From 10 to 20 c.c. should be obtained if possible, the centrifuge must be run from 5 to 10 minutes according to the speed obtainable and the fluid kept on ice and examined

Limited value.

Technique.

*Aqueous solution of sodium chloride 1% and potassium bichromate 5% (Determann).

within 24 hours. Examinations are best made by staining smear preparations as in the case of the blood. The triacid stain is satisfactory or Wright's stain may be used to save heating the specimens, though its methylene blue may over-stain in the sharply alkaline serum. Under the microscope we consider erythrocytes, endothelium and tumor cells as well as leucocytes and the following reasonably dependable conclusions may be drawn. (a). The presence of large *amounts of blood suggests carcinoma, tuberculosis, and the hemorrhagic diseases*, but may be present in cardiac and nephritic cases, though usually accidental. (b). In general *the predominance of polymorphonuclear forms indicates marked and relatively recent inflammation.* (c). *A large number of mononuclear cells indicates a less acute process, its later or chronic stages*, or, *the chronic stage of a non-specific exudate.* (d). *A large number of endothelial cells unassociated* with any considerable number of leucocytes speaks for a transudate. (e). *Specific tumor cells* may *if typically grouped prove a malignant growth*, but ordinarily can with no certainty be distinguished from single or grouped endothelial cells.

Diagnostic inferences.

Primary Tuberculosis is indicated by a predominance of lymphocytes after a week or ten days; *acute infection, by polymorphonuclear preponderance.* Predominance of endothelial cells in *tesselate* arrangement indicates a transudate. *Secondary tuberculous pleurisies* show polymorphonuclear predominance or a sediment of necrotic cells and debris. An exudate showing bacteria and 90% or more of polymorphonuclears means that empyema is imminent (Musgrave) *

INOSCOPY.—Jossuet's method of predigesting the coagulum of suspected exudates as preliminary to the examination for tubercle bacilli has proven of much value in pleuritic cases. Its findings have less accuracy, however, than animal inoculation and possibly than cytodiagnosis, so that it is the positive factor that should be considered, the failure of the test being no adequate proof of the absence of tuberculosis.

Useful, not certain.

Technique.—The fluid is collected in sterilized flasks and the clot when formed, thoroughly washed on sterile gauze spread over the mouth of a funnel or receptacle. The clot is then placed in a flask or flasks in each of which is poured from 20–30 c.c. of the solution (pepsin 2 gms., glycerin and strong HCl of each 10 c.c., sodium fluoride 3 gms.,

* Musgrave highly recommends Wright's stain, 3 parts, methyl alcohol, 1 part. Stain 20 to 45 minutes, treat with 8 or 10 drops of water, let it stand 2 or 3 minutes and wash gently by flooding with water several times, allowing it to stand a few seconds with each washing.

distilled water 2000). Digestion will be complete in about two or three hours at body temperature. Centrifugalization of the mixture for 10 minutes is followed by decantation and the staining of cover-slip preparations to which a bit of egg albumin may be added. Gabbett's method and stain should be used but the preparation must not be as vigorously decolorized as is usual in sputum mounts; 30 to 45 seconds being sufficient.

DISEASES OF THE BLOOD.

The Anæmias.

Types of Anæmia.—There are three principal types of anæmia, (1) *simple anæmia or chlorosis*, (2) *secondary anæmia*, which as its name implies is that which follows or is associated with other diseases. (3). *Pernicious anæmia.* In general one may say that *chlorosis* is characterized by a predominant hæmoglobin loss and a relatively slight diminution in the number of red cells present. That is to say the individual cell is poor in hæmoglobin. *Secondary anæmia* shows as a rule a more even reduction in both elements. *Pernicious anæmia* is the exact reverse of simple anæmia in that the cell decrease predominates and therefore the hæmoglobin value of the individual cell is high. (See Figs. 149, 158.)

Hæmoglobin-erythrocyte ratio.

General Considerations.—Color.—In the anæmias *pallor* as affecting the skin and mucous membranes is the rule, but is subject to frequent exceptions. *Persons may be pale, yet not anæmic, or ruddy and profoundly anæmic.* The author recalls two cases of advanced leukæmia in which the color was most deceptive. The student and the indoor worker is often pallid, yet not anæmic. The outward signs of anæmia should be sought for in the face, the conjunctiva, the lips, the mucous membrane, the lobes of the ear, the finger nails, tongue and pharynx. In *chlorosis* there is usually marked pallor and the skin is likely to show a greenish yellow tint. In *pernicious anæmia* there is ordinarily very marked pallor and the skin is of a lemon yellow tint. In *secondary anæmia* aside from pallor one meets with many variations in color. In acute *Bright's disease* the skin may be of a pasty white, or show a peculiar sallowness, brown or fawn color, more or less characteristic of the advanced chronic parenchymatous or "mixed" type of the disease. *Interstitial nephritis*, often yields no such evidence, being found in men of normal complexion, or in those who have the ruddy countenance of the high liver. *Syphilis.* Certain cases of congenital and tertiary syphilis show a peculiar sallowness

Misleading.

Variations.

impossible to describe, but easily recognized by those who have been shown it. *Malignant disease* especially carcinoma of the stomach is often associated with a somewhat characteristic earthy pallor. In diseases of the *mitral valve* one frequently finds anæmia masked by color due to chronic congestion.

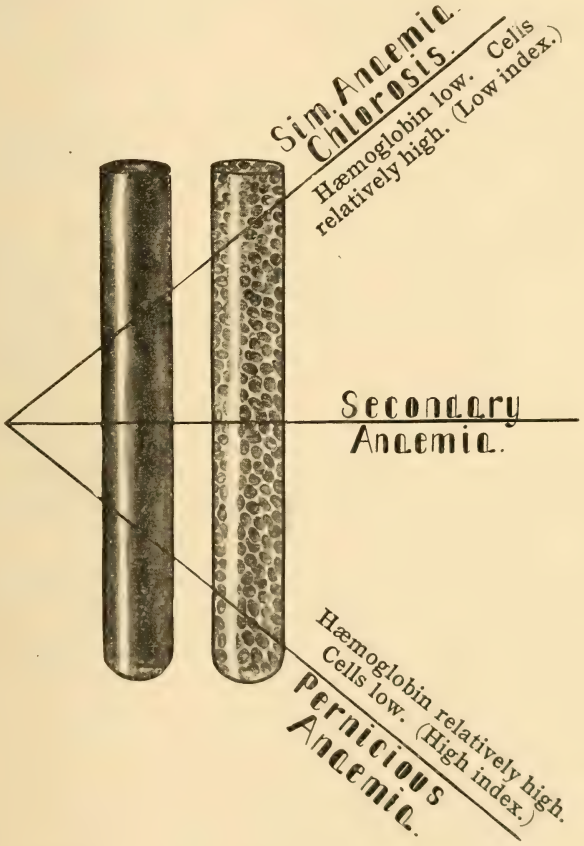


Fig. 158.

Muscular Weakness.—In all pronounced anæmias one meets with lassitude and muscular weakness, varying as a rule with the severity of the disease. It is least marked in mild anæmias of the secondary type and most evident in pernicious anæmia. **Temperature.**—Three-

fourths of all cases of pernicious anæmia are accompanied by temperature, usually of the intermittent type, with an evening exacerbation. It is by no means an infrequent symptom in *severe* cases of chlorosis* or secondary anæmia.

Gastro-intestinal Disturbances.—Symptoms referable to disturbed digestion, gastric or intestinal, are encountered chiefly in the chlorotic and pernicious forms of anæmia and in the leukæmias. *Anorexia, bulimia, nausea, vomiting, constipation and diarrhœa* are the common symptoms. Established and advanced *pernicious anæmia* is usually associated with exhausting attacks of diarrhœa and intercurrent vomiting.

Respiratory System.—*Dyspnœa* is more or less pronounced in all severe anæmias, but particularly so in marked chlorosis and pernicious anæmia.

Circulatory System.—*Palpitation, syncope and precordial oppression* are common. *Edema* of the extremities is not infrequent in chlorosis and is invariably present in *advanced* leukæmia and pernicious anæmia.

Bruit de Diable.

The pulse is usually of low tension, a curious humming sound is heard in the vessels of the neck, and various murmurs are heard over the heart and the great vessels.

Anæmic Murmurs.—The general characteristics of the murmurs of anæmia are:—(a). *Their systolic time.* (b). *Softness of quality.* (c). *Predilection for the pulmonary area.* (d). *Absence of marked and sequential cardiac outline changes.* (e). *Tendency to disappear as anæmia is improved by treatment.*

These murmurs may be heard over any of the valvular areas, *but are most frequent in the pulmonary and mitral regions.* A systolic murmur in the subclavian and carotid is a very common feature. *In certain cases where anæmia and neurasthenia are combined one may find outward symptoms suggesting aortic regurgitation viz.:—throbbing peripheral vessels, water hammer pulse, and even a capillary pulse.* This condition need not mislead one in the absence of a diastolic murmur,† and a modified aortic second sound.

Pseudo-Corrigan pulse.

Capillary pulse.

Nervous System.—The symptoms under this head are many and various. *Insomnia, drowsiness, vertigo, mental dulness and apathy, extreme irritability, perverted sensation, neuralgia, neurasthenia, hysteria,*

Anæmic neurasthenia.

* It nevertheless should always suggest tuberculosis in these cases and hence a thorough examination of the lungs.

† The author has reported one (terminal stage) case of pernicious anæmia in which all these symptoms were present without aortic valve changes adequate to explain the murmur, and a few other such clinical curiosities are to be found in the literature.

headache, and even delirium may be encountered though delirium is not often seen save in pernicious anæmia or the last stage of leukæmia. *Headache may be found in all degrees of severity in simple cases of chlorosis* and one form is peculiarly interesting by reason of its close resemblance to the headache of meningitis.* Such headaches are so severe as to require most radical measures for their relief, and often tend to recur until the general therapeutic indication, namely, the administration of iron, is fulfilled.

Misleading headache.

General Nutrition.—In *simple anæmia the fat is usually well preserved, giving* the patient a plump appearance. *In secondary anæmia the state of nutrition is dependent upon the primary lesion. In pernicious anæmia even in its later stage there is often an appearance of good nutrition,* due to the presence of overlying fat, *but the skin* in these cases *has a peculiar velvety feel, and the muscles are of the consistency of jelly to the touch.*

Varies with type of anæmia.

The Eye.—Muscular insufficiencies are common, *retinal hemorrhage frequently occurs in pernicious anæmia* and in all severe forms the ophthalmoscope shows a pale fundus.

Pale disc.

General Etiologic Factors.—*Age. Secondary and pernicious anæmia* occur for the most part in those at or above middle age. *Chlorosis* is essentially a disease of the earlier years, most often commencing at or about the age of puberty. *Sex.* 90% of all cases of *chlorosis* (simple anæmia) occur in young women. *Pernicious anæmia* is more frequent in the *male, secondary anæmia* follows the laws of incidence of the primary lesion.

Causes of Anæmia.—Little is positively known of the pathogenesis of anæmia and the scope and purpose of this volume forbids a discussion of the theories. *General Causes.* In connection with simple anæmia, one is likely to find defects in food, environment, habits and general sanitation. Boarding-school girls and working girls may suffer alike from insufficient or improperly prepared food, or from lack of variety, no less than from poor ventilation, overwork, or lack of fresh air or sunshine. Depressing emotions, such as grief, home-sickness or disappointment in love often play their part, and the effect of all of these general factors is shown in Arctic explorers who have in many instances suffered from severe forms of anæmia during the Arctic night, when nostalgia, darkness, fatigue, poor ventilation and improper diet were all present.

Bad hygiene.

Lovesickness and nostalgia.

*Two cases have come under the author's notice in which a mistaken diagnosis of cerebral meningitis had resulted from this curious condition.

Etiologic factors.

Secondary Anæmia.—This may be associated with the following conditions:—(a). *Repeated hemorrhages* (hemorrhoids, nose bleed, uterine hemorrhage, gastric ulcer, hepatic cirrhosis). (b). *Malignant* disease, especially carcinoma of the stomach. (c). *Syphilis*, leprosy and tuberculosis. (d). *Mineral poisoning*, arsenic and lead especially. (e). *Malaria*. (f). *Bright's disease* in its various forms. (g). *Intestinal parasites*. (h). *Auto-intoxication*. (i). *Any acute infectious disease*, acute rheumatism being the chief.

The foregoing statements as to general symptomatology are applicable to all forms of anæmia in varying degree. Considered as arbitrary types, *chlorosis stands at one pole, pernicious anæmia at the opposite, while secondary anæmias are ordinarily overshadowed and dominated by the symptoms of the causal lesion* and may assume varying and at times misleading forms.

CARDINAL SYMPTOMS OF THE ANÆMIAS.—Chlorosis.—*Age.* Young adults. *Sex.* Female in most instances. *Color.* Simple pallor or a "greenish yellow."

General symptoms as described in the preceding pages usually moderate in degree. See page 398.

Blood Findings.—*The color index is low*,* the hæmoglobin running from 12 to 75%, its average being about 45%. *Red cells are not proportionately decreased*, the average being 4,000,000 per c.cm., a count under 2,000,000 rare and *the average size of the cell less than normal:*

Achromia marked.

Stained specimens show marked *achromia* and old cases show marked *variation in the size* of the cells, but cell deformity, i.e. poikilocytosis, is rare. Nucleated red cells are seldom found and when present are normoblasts. The *leucocytes are normal or slightly decreased and relative lymphocytosis is common.* Myelocytes are rarely found and the eosinophiles are decreased. *The coagulation period is short. The patients appear as a rule, plump and well nourished* though pale, and their occupation is likely to be that of laundress, shopgirl, factory worker or school girl. *The developmental period is the one of commonest incidence.*

Unknown causation.

Pathogenesis of Chlorosis.—Practically nothing is known of the pathogenesis of this disease. It is commonly associated with constipation and gastro-intestinal derangement, and has given rise to the theory of auto-intoxication. Arterial hypoplasia has its advocates, and it has even been supposed to represent an incipient tuberculosis. No one of these theories can be accepted, entire, at the present time. *The*

* For definition of color index, see p. 378.

therapeutic test for chlorosis is found in the prompt response of its symptoms to the proper administration of iron.

PERNICIOUS ANÆMIA.—*Age.* Usually middle age. *Sex.* Males chiefly. *Therapeutic Test.* Iron fails absolutely. *Color.* Lemon yellow in most instances. *Nutrition.* External fat may be preserved until late, but tissues are flabby and gelatinous in feel.

Etiology and Pathogenesis.—Unknown. May be due to excessive hæmolysis or to deficient hæmogenesis. It follows, oftentimes, shock, mental strain, prolonged exposure and fatigue, syphilis, malaria, alcoholism, and in women repeated and excessive childbearing, but any or all of these factors seem to be only contributory. A mystery.

Symptoms.—As described under general symptomatology (p. 401), but presenting a most extreme type of muscular weakness, impaired nutrition, gastro-intestinal disturbance, vertigo, subcutaneous, mucous membrane, and retinal hemorrhage and slight edema of the extremities. The æsthenia is progressive, yet temporary recovery is common and may endure for several years though the ultimate prognosis is fatal.

Blood Findings.—The blood is pale and *slips* quickly off the ear or finger following puncture. The color index is *high* and the red cells are reduced on the average to 1,500,000 per c.cm., 1,000,000 being a common finding. The *hæmoglobin* may be as low as 15% but in most instances is *relatively* in excess. The *coagulation period* is prolonged, *the average diameter of the red cell is* increased, *poikilocytosis* or cell deformity is marked, the cells show great variation in size and *nucleated red cells* are invariably present though often found only after careful search and only rarely in great numbers. *The megaloblasts must be found to outnumber the normoblasts or a positive diagnosis is not justified.* An excess of microblasts or of normoblasts usually indicates a severe secondary anæmia. *Polychromatophilia*, i.e. irregularity in staining and consequent lack of uniform color in certain cells is a marked characteristic of this disease. *The leucocytes are diminished in ¾ of all cases and* this leukopænia may be extreme, the average being less than ½ the normal count. *Myelocytes* are *nearly always present though in small numbers.* It is usually possible to make an accurate diagnosis of pernicious anæmia by the blood findings alone but now and then cases occur in which the clinical history and physical signs must be invoked. *Bothriocephalus infection and nitro-benzol poisoning present an almost identical picture. Advanced*

High color index, and low erythrocyte count.

Predominance of megaloblasts.

Its simulators.

cases of malignant disease of the stomach may present quite as extreme an anæmia, but in this *the small cell type usually predominates, both as to nucleated and non-nucleated cells.*

SPLENIC ANÆMIA.—This condition has not yet made itself a place in medicine as a clinical entity and its features do not seem to be sufficiently characteristic to constitute a separate disease. *Anæmia with splenic enlargement* would seem to be the more correct term. *The one constant* blood finding *is leukopœnia*, the average leucocyte count being about 50% normal. So far as hæmoglobin and erythrocytes are concerned any form of anæmia may be simulated, the marked secondary type being that commonly shown. *We must regard the condition for the present as an interesting symptom-complex, lacking the glandular enlargement of Hodgkin's disease and the peculiar blood changes of leukæmia, and adding to the ordinary symptoms of anæmia that of splenic hypertrophy, commonly associated with hepatic enlargement.* A large number of these cases prove to be malignant growths on autopsy.

Only a syndrome.

LEUKÆMIA.—(White blood). *Historic Note.* Until 1841, this disease was regarded as a purulent inflammation of the blood. John Hughes Bennett and Virchow gave the first accurate account of the disease.

Pathogenesis.—Leukæmia is undoubtedly a primary affection of the bone marrow and the lymphoid structures, the essential change being one of myelocytic or lymphocytic hyperplasia, leading to deficient red cell production.

Varieties.—These are two forms of leukæmia. (a). *Spleno-medullary.* (b). *Lymphatic.* In the first form the blood assumes the so-called *myeloid* type, in the second, the *lymphoid.* Although several cases of acute myelogenous leukæmia have been reported, we may for the present regard it as essentially a chronic disease. Lymphatic leukæmia on the other hand may be either acute, subacute or chronic.

Essentially chronic.

SPLENO-MEDULLARY LEUKÆMIA.—Etiology.—*Age.* May occur at any age, but chiefly in middle age. *Sex.* Males for the most part. Other etiologic factors are not well understood.

Symptoms.—The disease is not likely to be detected until well advanced, and the general symptoms are then those of a profound anæmia with an especial tendency to hemorrhage and serous effusions. Dyspnœa is marked, loss of weight is usually extreme and both liver and spleen are enlarged, the latter to an enormous degree

in certain cases, and *always* to a marked degree.* It is astonishing that so great an enlargement may be unattended by serious discomfort, but such is oftentimes the case. Insidious in development.

Blood Findings.—The red cells are markedly reduced and the *white cells are greatly increased* in number, the average count being 400,000, and the maximum frequently exceeding 1,000,000 cells. The characteristic white cell of leukæmia is the myelocyte and from 20% to 50% of the leucocytes present are of the myelocytic type (see plate II. The *eosinophiles* are also present in unusual numbers, Myelæmia.

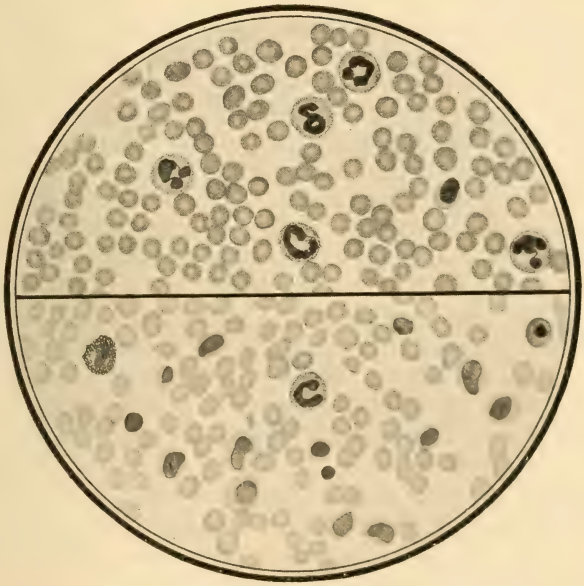


Fig. 159.—Lower section shows a lymphatic leukæmia. Upper section normal blood showing leucocytosis. The types of cells should be carefully noted.

showing both an actual and relative increase. Both the *lymphocytes and polymorphonuclear cells* though absolutely increased are relatively diminished. *Megaloblasts, normoblasts and microblasts* are present in quantity and their nuclei frequently show mitotic changes. *One of the most striking though not invariable symptoms of the later stages*

*In one case shown by the author to the Minnesota State Medical Society in 1903, the splenic margin actually touched the anterior superior spine of the right side.

Hemorrhagic tendency.

of the disease is the tendency to hemorrhage from mucous membranes. These are often large, persistently recurrent, and demand that every patient should be duly warned and instructed concerning measures for their relief or control. They often prove a terminal event, or death may result from esthenia, or either gradual or sudden cardiac failure often associated with general dropsy.

LYMPHATIC LEUKÆMIA.—The essential features of lymphatic leukæmia are two in number. (a). *Enlargement of the lymphatic glands.* (b). *An absolute lymphocytosis in which either the large or small cell type may predominate.* The chronic cases last for years, the acute cases may terminate in a month or six weeks.

Lymphocytosis absolute.

Lymphomata.

Symptoms.—Any or all groups of lymphatic glands may be enlarged, the *tumors are as a rule separate and movable, free from active inflammatory changes and there is little or no tendency to suppuration.* The spleen may be moderately enlarged *but the glandular symptoms predominate. Fever* is present in variable degree in all acute cases and the later stages of the chronic form. *Pressure symptoms* may be marked and in three of the author's cases death occurred from suffocation due to the pressure of mediastinal growths.

Blood Findings.—*Nucleated red cells are rare as compared with the myelogenous leukæmias,* though in the acute type of the disease they may be present in considerable numbers and the same is true of certain of the leukæmias of children; ordinarily, however, *the normoblast predominates.* The leucocyte increase while striking *is seldom as large as in leukæmia of the myelogenous type,* the average lying between 200,000 and 250,000 cells, the maximum being about 1,000,000, but in a case observed by the author the count reached 2,133,000 for the single examination possible and was probably a preagonal increase as it occurred but 24 hours before death. The remarkable predominance of the lymphocytic type (average 90%) is striking and the cells show many atypical forms. The myelocytes are relatively rare, eosinophiles are diminished and neither the mast cell nor the basophile are common.

A remarkable case.

Differential Diagnosis of the Anæmias.—*The enormous value of blood examinations is evident if one considers the difficulties surrounding the purely clinical differentiation of the conditions described as compared with the ease with which each disease may be identified by hæmatologic methods.* As regards *anæmia in any of its three chief* forms and leukæmia *no confusion is possible* and *the two chief varieties of leukæmia are quite as sharply contrasted.* The glandular enlargement of *Hodgkin's disease, tuberculosis and syphilis cannot give the slightest difficulty*

Blood findings, the only means.

if the blood findings are invoked, *nor can the various enlargements of the spleen be confused with myelogenous or lymphatic leukæmia.* Certain cases of profound secondary anæmia, particularly those associated with carcinoma of the stomach or atrophy of the gastric tubules (not merely the simple *achylia gastrica*) may at times present a picture almost indistinguishable from that of pernicious anæmia and to these may be added bothriocephalus anæmias which are probably symptomatically identical with pernicious anæmia proper. It is probable that in gastric atrophy the blood findings are always those of the profound secondary type.

Subdivisions and Transition Forms.—It is quite possible that further subdivisions of leukæmia may be made in the future, and indeed many cases of variation in the relative predominance of cell types and apparent transition, are constantly being placed on record.* *Von Jaksch* has reported a *multiple periostitis* with splenomegaly and myelocytic anæmia, multiple arthritis, profuse sweats and the same or an even greater tendency to the hemorrhages characterizing the later stages of myelogenous leukæmia. In *chloroma* the blood findings may be indistinguishable from those of lymphatic leukæmia but the clinical symptoms are strikingly different. *Still* has reported a syndrome, the main feature of which is an *infantile arthritis* with enlargement of the lymph glands and spleen, but lacking the blood findings of leukæmia and presenting usually the history of rachitis.

Still's syndrome.

Prognosis.—In anæmias of the *chlorotic* type the prognosis is always good unless it be a part of the clinical picture of gastric ulcer or incipient tuberculosis. *Secondary anæmias* are wholly dependent upon the possibility of removing the primary cause. *Pernicious anæmia* is probably invariably fatal, though long periods of apparent recovery may be noted.†

As regards *leukæmia*, there is an invariably fatal termination though of late cures have been credited to the X-Ray treatment. It is probable that these cases will also prove fatal after a period of apparent arrest.

POLYCYTHEMIA.—A more or less marked increase of erythrocytes above the normal count is observed in a great variety of conditions, some important, others trivial, and curiously enough the permanency of such a condition still remains unproven. It

Not uncommon.

* The author has observed a case of apparently typical Hodgkin's disease become in all respects lymphatic leukæmia.

† The longest duration of such a period of false cure personally observed was five years, a second period of about one year followed, succeeded by a rapidly fatal relapse.

High altitudes.

usually represents nothing more than blood concentration or stasis and in high altitudes is perfectly accounted for by the assumption that nature meets an actual need associated with the rare air and the dyspnœa first produced in a non habituated person. This assumption is borne out by the secondary partial reduction noticed in those who take up permanent residence. A similar increase may follow baths, hot or cold, massage, violent exercise and the administration of certain drugs or may be associated with the process of digestion, blood regeneration, vomiting, profuse sweating, the removal of exudates, profuse diarrhœas, and, it is said, with myxœdema and acute yellow atrophy. Its presence in connection with organic heart disease, emphysema, stenotic dyspnœa and similar conditions affecting the circulation is readily understood.

Multiple causes.

OSLER'S DISEASE.—(Cyanotic or "Cryptogenetic" Polycythemia.) This peculiar disease or syndrome comprises:—(a). *Marked increase of the red cells.* (b). *Cyanosis.* (c). *Enlargement of the spleen.* (d). Frequently, *hepatic enlargement.* (e). Commonly, *albuminuria.* (f). *Hyperviscosity of the blood.* (g). *Rapid coagulation.* The disease was first noted by Saundby and Russell* and by McKeen† in 1901, and in 1903 Osler's report of cases and masterly description brought the condition into prominence. In Cabot's‡ series three of the four cases are peculiarly interesting because of the lack of constant relation between the erythrocyte and leucocyte count and the entire failure of correspondence in either with the hæmoglobin values. Hæmoglobin values of 165 and even 200 have been reported but in one of Cabot's cases it was but 85 with a count of 8,484,000 reds and 15,000 whites, and in another of his cases it was 105 with a count of 11,352,000 reds. Moderate or absent leucocytosis is the rule. In reporting cases the *chief clinical factors* must be demanded as a polycythemia of marked degree may co-exist with cyanosis under other conditions.

A recent discovery.

HODGKIN'S DISEASE.—(Pseudo-leukæmia, lympho-sarcomatosis, lymphadenosis, anæmia lymphatica, adenie, lymphadenie, etc.)

Definition.—A disease characterized by hyperplasia of the lymph glands, with or without splenic enlargement (75%), and varying degrees of anæmia.

Historic Note.—The disease was first reported by Malpighi in 1669, who did not recognize it as a distinct disease. Craigie, in 1828, differentiated the anatomical character of the glands from those of carcinoma

* Lancet, 1901, Vol. 1.
† Boston Medical and Surgical Journal, June 20, 1901.
‡ Clinical Examination of the Blood, p. 71, 1904.

and tuberculosis, but to Dr. Hodgkin (1832) we owe our first definite description of the disease as a clinical entity. It did not, however, attain general recognition until the sixth decade of the last century.

Etiology.—The acute forms strongly suggest a definite infection, and by some this is believed to be tuberculous in its nature. The theory, however, has no substantial or well proven foundation, and the true cause remains unknown.

Pathology and Morbid Anatomy.—The essential change is one of hyperplasia of the lymph glands involving primarily limited areas, but tending to extend widely. The cervical and inguinal glands are usually the first affected, the former giving to the disease its most marked characteristic. The tissues of the neck may entirely lose their normal outline, and the glands there and elsewhere tend to steadily enlarge without marked tenderness or symptoms of inflammatory change. They tend to maintain their individual outline until late in the disease, and the skin usually remains unaffected and unattached to the subjacent tumors. When periadenitis occurs in the later stages lobulate tumors are formed which may lose the firmness characteristic of the earlier stages and become softened and rarely, may undergo ulceration. The deep glands above the trachea, those of the larger bronchi, of the mediastinum and retroperitoneal area become involved and in some instances form large tumors and greatly complicate the case. The spleen is enlarged in 75% of the cases and shows overgrowth of the lymphoid bodies. The marrow of the long bone may present much the same appearance as in spleno-medullary leukæmia, the liver and kidneys are likely to contain lymphoid tumors and both the lymph nodes and the bone marrow contain an excess of eosinophile cells. Microscopically the lesions show a proliferation of endothelial cells, the formation of giant-cells, increase in connective tissue, and overgrowth of the lymph nodes.

Lymphomata without blood changes.

The Blood.—There are no changes in the blood peculiar to this disease, the condition being one of secondary anæmia.

Acute Hodgkin's Disease.—Certain cases develop so rapidly as to be properly called acute, and may run their course to a fatal termination in a few weeks. The symptoms are, however, not different from those of the chronic type, save that there is a certain concentration of all clinical phenomena.

Symptoms.—The onset is insidious, save in acute cases, and the enlarged glands, usually in the cervical region, but sometimes in the groin and axilla, may first attract attention. The glandular enlarge-

Chronic form.

ment may primarily be either unilateral or bilateral, but extends and tends to become general. The changes in the neck and the peculiar outline produced have given rise to the descriptive term "the Hodgkin's collar." As the disease progresses, though often only after several months, a considerable degree of secondary anæmia, and more or less progressive emaciation appear. If, as is not infrequent, the mediastinal glands take on a rapid growth, the pressure symptoms may be extreme and will precisely resemble those seen in any other mediastinal tumors (see p. 205), pain, cough and dyspnœa, paroxysmal or continuous, being the chief symptoms. Fever is present in the advanced cases, and is usually of a mild hectic type. Splenic enlargement is readily made out in three-fourths of all cases.

The Hodgkin's collar.

Differential Diagnosis.—From *tuberculous adenitis* it is usually readily differentiated by the absence of marked inflammatory symptoms, a history of tuberculosis, past or present, by the excision, if necessary, of a small gland and its examination, and by the fact that tuberculosis is more generally unilateral, and that its tumors never attain a large size without marked evidence of inflammation and caseation. From *leukæmia* it is absolutely differentiated by the negative blood examination (see leukæmia, page 404). *Syphilis* must be differentiated by the history and presence of scars, and the difference in the character and extent of the glandular swelling, which in this disease, though more or less general does not often attain large size. True sarcoma tends to spread to other tissues. *Simple lymphoma* is localized and lacks the predominating symptoms of Hodgkin's disease.

Unusual Symptoms.—An enormous number of symptoms might be named in connection with the development of glandular masses in the abdomen, amongst these being bronzing which results probably from pressure upon the solar plexus. One has only to bear in mind that large tumors may develop in remote regions producing exactly the same effects as would any other tumors in the same location.

Bronzing possible.

Prognosis.—The disease is almost invariably fatal, its course occupying usually one or two years. Misleading periods of improvement followed by relapse are extremely common and this applies to both the general and local manifestations of the disease.

PURPURA.—(Morbus maculosus.) **Definition.**—A morbid state characterized by subcutaneous hemorrhages with or without fever and other constitutional symptoms.

Comment.—So ill-defined and little understood is this disease that an arbitrary classification serves the purpose of clinical convenience and

little more. It seems to bear a close resemblance to certain forms of urticaria, scurvy and hæmophilia, and possesses but two constant and invariable symptoms, viz.:—Subcutaneous hemorrhages, and a prolonged blood coagulation period. Cases may be grouped under two general heads:—(1). **Complicating purpuras,** viz.:—those occurring in connection with (a) acute infections, (b) chronic diseases associated with cachexia. When purpura occurs in association with acute infection it ordinarily indicates an exceptionally severe type of the disease. One may encounter it in typhoid fever, smallpox, malignant endocarditis, septicæmia, pyæmia, whooping cough and measles as well as in the more virulent of the tropical diseases. In cerebro-spinal meningitis and typhus fever it represents the usual exanthem. Of the chronic diseases it is seen most frequently in connection with scurvy, pernicious anæmia and leukæmia, and not infrequently in tuberculosis, Bright's disease, Hodgkin's disease, syphilis, and in connection with various cardiac lesions, particularly those associated with marked degeneration of the myocardium. In epilepsy it is not uncommon and sometimes points the way to a correct diagnosis in cases of an obscure nocturnal type. In such instances it is usually slight and transient, often showing only on the face as an indistinct capillary streaking, which may last for a few hours or a few days. In hysteria, the same form may be present, or the rarer forms amongst which the most curious is the cruciform purpura in which the hemorrhages appear at points corresponding to the wounds of the Saviour on the Cross. It may be seen at once that in epilepsy, whooping cough and chronic heart disease mechanical strain plays a great part. (2). **Arthritic Purpuras.**—In the three forms of purpura coming under this class there is a joint involvement distinctly rheumatic in type. Under this we have:—(a). *Simple purpura*.. (b). *Peliosis rheumatica* (Schönlein's disease). (c). *Henoch's purpura.* This classification need not be confusing if the following points are borne in mind: in all, the essential lesion is hemorrhage, all are accompanied by fever and by joint lesions, but:—**Simple purpura** is of a *mild type and short duration*. It has no other distinguishing features. **Peliosis rheumatica** is much more severe with *higher fever, more marked joint involvement sore throat* usually at the onset and not only *marked purpura*, but frequently pemphigoid spots, purpuric edema and marked urticarial manifestations of the ordinary or giant variety. *The distinctive feature, however, is the involvment of joints to a marked degree.* **Henoch's Purpura.**—Differs from the two already named, primarily, in the fact

Significance of purpuric exanthem in acute disease.

Cruciform purpura.

Hemorrhage the essential lesion.

that *gastro-intestinal symptoms such as nausea, vomiting, diarrhœa, and hemorrhage from mucous membranes* are the predominating features, *while arthritis is usually slight.* In all other respects it may closely resemble the other forms.

Purpura Hemorrhagica.—(Morbus Werlhofii). This terrible disease may be rapidly fatal, and *is characterized by severe and often intractable hemorrhage from any or all mucous membranes, associated with high fever.*

Differentiation possible.

It will be seen from the foregoing description that though the basic symptom is the same in each and every variety the differences presented are such as to permit of a clinical differentiation in most instances.

HEMOPHILIA.—(*Aima,* blood, *phinein,* love). **Definition.**—A morbid state characterized by an extraordinary tendency to spontaneous or induced hemorrhage and an especial predilection for young males in succeeding generations.

Vague theories.

Etiology.—The actual cause is unknown; deficiency of fibrin is evident; and a peculiar thinness of the vessel walls, a neuropathic tendency and abnormal alkalinity of the blood have been advanced as unproven theories of causation. *Age and Sex.* It affects chiefly male children under ten years of age, cases being rare in the later years of life, and usually less severe in their nature. **Heredity.**—The disease furnishes one of the most interesting known examples of atavism; affecting male children through maternal heredity, the girls of a family seldom showing any hæmophilic tendencies, yet passing it on to their male children. In one family in Switzerland, the disease can be traced back over 300 years.

Atavism.

Beware of "bleeders."

Symptoms.—*Intractable nose bleed is most common, but hemorrhage may occur from the lips, gums, throat, stomach, urethra, lungs, bowels or in the female from the uterine mucous membrane,* though strangely enough labor is not ordinarily attended with extraordinary hemorrhage. *No operation should be undertaken without some inquiry as to this condition,* as information upon the subject is seldom volunteered by members of these foredoomed families and slight wounds or bruises, even the puncture of the ear or finger for blood examination, the drawing of a tooth, the application of a leech or a vaccination, may bring about a fatal hemorrhage, and *more extraordinary still is the fact that spontaneous hemorrhage from mucous membranes, the free skin (lobe of ear, tip of finger), may come on without any apparent excitign cause.* The mortality is extremely heavy, and pseudo-rheumatic affec-

tions are common, as are ecchymoses and hematomata, these showing its close resemblance to purpura and scorbutus.

Prognosis.—A majority of male children of affected families die under ten years of age, though light cases occur in which after the age of puberty the tendency is lost.

SCORBUTUS.—(Scurvy). A morbid state peculiarly dependent upon diet* characterized by profound weakness, anæmia, and a marked tendency to hemorrhage from the gums.

Etiology.—The actual cause is unknown, but the conditions of its development are well understood. They are (1). *A diet from which fresh vegetables, fruits and meats are excluded.* (2). *Bad air, over-crowding, over-work, deficient light.* (3). *Mental depression, is present in most cases.* Up to within the last century scurvy was the scourge of seamen and of armies. During the Crimean war, there were 23,000 cases in the French army alone. Military and naval expeditions had sometimes to be abandoned on account of outbreaks of the disease and East Indian ships frequently lost half of their crews in one voyage. In one of the early Arctic expeditions 50% of the members died of the disease. In a recent Antarctic expedition the effects of the factors mentioned above were clearly shown, though a better food supply prevented a general epidemic. Essentially, however, scurvy belongs to generations past.

Once a scourge.

Symptoms.—The *gums are spongy and bleeding, often ulcerating, purpuric symptoms are pronounced, areas of brawny induration appear,* especially in the lower extremities, *subperiosteal hemorrhages* may occur, especially in children, and the swelling is commonly over the outer aspect of the femur, but occasionally over the tibia and is in every case hard, tense and exquisitely tender. Epiphyseal suppuration may occur and in all cases hemorrhages into the joints and retina are not uncommon. Physical weakness is extreme, there is pronounced and progressive mental depression and usually edema of the ankles. As occurring in children, scurvy is known as *Barlow's disease,* having been described by Drs. Cheadle and Barlow, in 1878. As before stated, the most suggestive symptom in children is the subperiosteal extravasation. The child lies motionless, shrinks from the touch, and presents what is really a characteristic swelling, the author having seen, as a student, in Dr. Barlow's and Mr. Edmund Owen's wards an immediate diagnosis on sight and touch in many instances. The disease is never

In children.

*In a *typical* adult case recently observed by the author absolutely no connection with diet could be traced.

seen in breast fed children and occurs usually from the sixth to the sixteenth month. It derives its general interest from the fact that the other symptoms of scurvy as seen in the adult may be entirely lacking.

ADDISON'S DISEASE.—(Morbus Addisonii). This was first reported by Dr. Addison of Guy's Hospital, in 1855, and in his monograph he attributed the ailment to a disease of the suprarenal capsules.

Peculiar syndrome.

Definition.—A disease characterized by *progressive weakness, gastro-intestinal disorders and a peculiar pigmentation of the skin and mucous membrane.*

Etiology.—The exact nature of the causative factors remains as yet undetermined. An antecedent tuberculosis seems the most important. Various diseases may precede it but their direct connection in causation is unproven. Poor food, insanitation, excessive mental or physical labor are often associated factors, but may be only coincident. Males are affected much more frequently than females (60–70%). It is most common in middle age, yet cases have been reported in infants. *Nearly 90% of these cases show demonstrable lesions of the suprarenal capsule, and in 80% of these instances the lesion is clearly tuberculous. In a relatively small percentage a lesion of the capsule is not evident, and furthermore, the suprarenals may be removed by operation or be congenitally absent without of necessity producing the disease.* Its relation to lesions of the abdominal sympathetic, demonstrable or otherwise, seems probable.

Etiologic paradox.

Morbid Anatomy and Pathology.—Any one of several conditions may be found at autopsy, These are, atrophy, simple or due to chronic interstital inflammation, malignant disease of the capsule, extravasation of blood, inflammation or pressure affecting the semilunar ganglia and in a vast majority of cases tuberculous infection of the suprarenal itself. It seems probable that Addison's original theory, that the *disease was due to a loss of function on the part of the adrenals is correct,* though this cannot at present be absolutely proven. The involvement of the semilunar ganglia may materially affect the secretions of these glands and contribute to the symptom complex. This view seems on the whole more rational than that advanced by some observers, which holds the nervous system entirely responsible for the disease.

Lost adrenal function.

Symptoms—The disease is *insidious* in its approach; *is rarely suspected until pigmentation* becomes well marked; the *gastro-intestinal disturbance* as well as *the loss of strength* tends to increase, yet there are cases showing long periods of latency and relative immunity from *vomiting, nausea and diarrhœa.* A slight secondary anæmia is usually

present, but is not an important factor. As the disease progresses, *attacks of syncope, vertigo* and *palpitation* may be troublesome. *Vomiting may become persistent,* and *indeed uncontrollable,* sometimes ending fatally, and again subsiding after several days. *A history of tuberculosis, past or present,* in some other part of the body is often obtainable. *The pigmentation of the skin* is very variable in its intensity, the typical case showing a deep bronze color that is quite unmistakable though the early pigmentation may be little more than a brownish yellow or definite brown, and, rarely, the case may die before this deepens to the typical shade. It shows certain interesting and important characteristics as regards distribution, in that *it is most marked in the portions of the body exposed to light, such as the face and hands,* and to those exposed to *pressure or friction from clothing,* being emphasized in the axilla, groin, under the breasts, or at the waist line and knees. Areas of pigment atrophy often appear making a sharp contrast and the *mucous membranes of the lip and buccal surfaces* especially are likely to show *patchy* pigmentation.

Pigment distribution.

Mucous membranes.

Diagnosis.—The diagnosis is ordinarily easy in view of the grouping of progressive loss of strength, marked gastro-intestinal disorders and the peculiar pigmentation as above described.

Differential Diagnosis.—Verminous bronzing is rarely seen except in the clinics of the great medical centres, being due to the constant presence of vermin attended by scratching, which results in a more or less profuse pigmentation; in most instances the attending signs would be lacking as would certainly be the mucous membrane pigmentation.* *Cirrhosis of the Liver.* In a few instances hepatic cirrhosis in its last stages may produce a marked pigmentation, but in such instances there is almost always abundant evidence that the color is due to an actual jaundice. In no such case in the author's experience has any such doubt arisen. *Argyria* (chronic silver poisoning). Is excessively rare at the present day, and the color is quite different from the pigmentation of Addison's disease, being more distinctly gray, and when excessive, showing a distinct bluish black tint; moreover, it is ordinarily limited to the face and hands, and the associated symptoms of the latter disease are absent. Many diseases are accompanied by pigmentation and peculiar patchy discoloration, i.e. malignant disease, involving the abdominal organs, certain cases of tuberculosis, of chronic nephritis, etc., but it does not seem that the difficulties in

"Vagabond's disease."

Jaundice, not pigmentation.

* In such cases as have been observed by the author, the likeness was only sufficient to drawing a passing reference.

diagnosis are so great as to permit us to accept the dictum of Leube, to the effect that one "should forego any diagnosis of affections of the adrenal bodies." The tuberculin reaction sometimes offers some confirmatory evidence.

Tuberculin test.

Variable.

Prognosis.—The disease is almost invariably fatal, usually within one or two years, the last case observed by the author died after an illness of only a few months' duration. In rare instances life is prolonged for more than a decade.

LYMPHATISM.—(Status lymphaticus, constitutio lymphatica). **Definition.**—A morbid state characterized by coincident hyperplasia of the lymph nodes, spleen, thymus, lymphoid bone marrow and in hypoplasia of the heart and arteries and showing a decided tendency to sudden death.

Historical Note.—The discussion of this curious condition is really a revival of ancient knowledge. Nearly three centuries ago *the association of an enlarged thymus with sudden death* is said to have been noticed by Plater, and the *thymic asthma* was described by Kopp in 1830. This he regarded as a pressure asthma, later discussions occurred, when the subject was again dropped to be renewed during the past few years.

"Thymus death."

Etiology.—Little is known of the etiological factors, but the disease is one of youth and early childhood.

Symptoms.—The symptoms upon which a diagnosis must be based are essentially pale, pasty, rachitic or tuberculous aspect, *enlargement of the spleen and of the superficial glands*, usually associated with *tonsillar hypertrophy*, adenoids and *dulness over the sternum*. The *thyroid* may be slightly enlarged, and there may be evidence of the involvement of the *mesenteric glands*. The chief importance of the disease lies in its relationship to early and sudden deaths in the young, both as occurring without apparent cause and in connection with operative or therapeutic procedures, the so-called "*thymus death*." This assumes considerable importance to both the physician and surgeon, cases *having been reported as following adenoid operations, anæsthesia, administration of antitoxin, convalescence from various infections, and croup*. Paltauf ascribes to this condition many of the cases of sudden death in children. The condition merits further study and observation, and its symptomatology should be kept clearly in mind. *These cases will usually show a relative but not an absolute lymphocytosis.*

Relative lymphocytosis.

MYXŒDEMA.—(Cretinism, cretinoid change, etc.) **Definition.**—A chronic functional disorder of cellular metabolism associated

Athyrea.

with structural changes and impaired function on the part of the thyroid gland.

Classification.—Although lacking in essential differences, three groups may be distinguished. First, *cretinism* or myxœdematous changes in the infant or young child. Second, *myxœdema proper* as it appears in the adult. Three, *operative myxœdema*, which covers the changes attending the complete removal of the thyroid gland.

Cachexia strumipriva.

Fig. 160.—Cretinism. (Courtesy of Dr. Henry Jackson.)

Symptoms of Myxœdematous Change.—The symptoms are essentially alike for all three groups showing myxœdematous changes.

Etiology.—That loss of function usually but not invariably associated with atrophy of the thyroid gland is directly responsible for myxœdematous changes has been well established by both clinical and experimental observations. The disease attacks women five times more often than men, is peculiarly frequent in certain countries and districts, and in the case of cretinism seems to be markedly hereditary; the offspring of people who have removed from regions where cretinism is endemic showing a marked tendency to the disease whatever the nature of their new environment Congenital syphilis, a tubercular taint and rachitis seem to favor the development of the disease in children and infants. In these the disease is ordinarily directly due to congenital atrophy or deficient function, but does not make its appearance for several months after birth as a rule, and sometimes not for many years.

Symptoms of Cretinism.—The appearance of the affected child

The cretin. is characteristic and unmistakable. The *face is senile, coarse and stupid,* the *lips and eyelids are thickened* and overgrown, *tongue often protrudes* from the mouth, the *root of the broad, flattened nose is depressed,* the *head is large, clumsy and rests upon a thick, short neck,* the *stature is dwarfed,* and a child of 14 or 15 appears often to be but 2 or 3 years old. The *legs are bowed,* the *abdomen is prominent,* the *joints are thick and clumsy,* the *gait is awkward,* dragging, laborious and *marked lordosis* is often present. The *skin is sallow, the hair is often thin and brittle,* the *skin usually dry* though it may *be greasy,* and *signs of arrested mental development* are as striking as the physical appearance, the condition being usually distinguishable from other forms of idiocy. The *fontanels remain open* and *muscular weakness* is usually marked. Cretinism may be endemic or sporadic, and a peculiar form described is the result of an atrophy of the thyroid with an acute febrile disease.

Arrested development.

Symptoms of Myxœdema in the Adult.—The *skin* is *dry, harsh and pasty,* the *hair is lustreless* and *brittle,* the *face is peculiarly moon shaped* and *lacking in expression.* The *features are clumsy and coarse,* the *nostrils broad, lips thickened* and *mouth enlarged, both thought and motion is slow and clumsy, the whole body shows an increase in bulk, and the superficial tissues have an appearance like edema, yet show* no *pitting on pressure. Large pads* of firm inelastic tissue usually *appear above the clavicles,* and the *hands and feet are pudgy and clumsy. Headache* may or may not be present, *irritability* is usually marked, and *actual delirium, hallucination* and *even true insanity* may develop. The function of the organs in general is normal and though albuminuria and glycosuria have been reported they are probably merely an accidental association. *The course of the disease* may extend over a period of from 10 to 20 years, the patient dying of intercurrent disease. Osler reports what is apparently a case of acute myxœdema and in some cases it seems to have been associated with the development of exophthalmic goitre. The author has reported a case in which symptoms of myxœdema co-existed with those of acromegaly.*

Acute myxœdema.

Operative Myxœdema.—Does not differ materially from that described above. This condition is rare because it necessitates for its development the complete or almost complete removal of the thyroid gland.

Diagnosis of Myxœdema.—In the adult this must depend upon the *peculiar color,* marked edematous aspect of the body, and *the failure of the bulky tissues to pit on pressure,* combined with the *physiognomy*

*See Transactions of the Association of American Physicians, 1905.

and *mental state* of the patient. Osler properly warns the clinician against laying undue stress upon the supraclavicular pads. As a matter of fact, this condition is very commonly seen in any stout person otherwise in perfect health. The improvement *of all symptoms* under the administration of thyroid gland offers conclusive proof of the nature of the disease but *loss of bulk alone is not sufficient evidence.*

Insufficient evidence.

EXOPHTHALMIC GOITRE.—Definition.—A disease characterized by prominence of the eyeballs, rapid pulse, enlargement of the thyroid gland and fine tremor associated with marked nervous manifestations. It may assume an acute, subacute, or chronic form, the last being the one nearly always encountered. The names of Basedow and Graves have apparently been improperly used in connection with this disease, and it should be called Parry's disease, as he described it quite fully in 1825, indeed having made notes of a case as early as 1786, half a century before the description of Basedow and Graves appeared. (Osler.)

Parry's, Graves', Basedow's or Flajani's disease?

Etiology.—The disease is due to disturbed thyroid function (probably hypersecretion), the exact nature of which is unknown. It is rare in men, quite common in women, and appears usually between the ages of 20 and 40. It seems to be well established that violent or depressing emotion even though transient may produce it and in some instances it has seemed to the author that there was a marked sexual element. Despite its marked nervous symptoms it is probably not properly classified as a disease of the nervous system but rather of the thyroid gland itself.

Hyperthyrea.

Symptoms.—Various authors have reported *acute cases* presenting the cardinal symptoms with marked gastro-intestinal disturbances and profound and progressive cardiac weakness, i.e. precisely the same phenomena that may bring a chronic case to a fatal end. Ordinarily, the onset of the disease is gradual and progressively slow, its tendency being towards increase in the severity of all symptoms but usually with marked recession periods if under treatment.

Acute cases.

Thyroid Enlargement.—It usually involves both lobes, but not equally and may be unilateral. Venous bruit and loud systolic or double murmurs are commonly heard, there is usually a palpable thrill and often a visible pulsation in which the whole gland may participate.

Usual second symptom to appear.

Exophthalmos.—This protrusion of the eyeballs varies greatly in degree, and it is said may become so great as to actually dislocate the eye.* The change is almost invariably bilateral, the vision is seldom

*No such extreme cases have ever been observed by the author.

affected, but much stress has been laid upon three so-called *signs* in connection with *exophthalmos*. These are:—(1). The failure of the lid to follow the downward movement of the eyeball (Graefe's sign). (2). Spasm or retraction of the upper lid (Stelwag's sign), and deficient convergence of the eyes (Moebius' sign). *The first two are practically useless* on account of the rarity with which they are observed and all three are somewhat superfluous clinically. The characteristic associated appearance in any considerable grade of exophthalmos is the rim of white which appears between the corneal margin and both lower and upper lids but this is not necessarily due to either spasm or lid retraction.

Over-rated signs.

Rapid Pulse.—This is often the first symptom to attract the attention and the pulse rate may vary from 90 to an uncountable beat, as is frequently seen in fatal cases. The pulse may be regular or irregular; peripheral pulsation is marked and a capillary pulse is often present as is the venous pulse. All sorts of bruits may be heard at the base and over the gland or the vessels of the neck, and a systolic murmur is seldom lacking in the mitral area. Such murmurs are sometimes autoaudible, or to be heard at a distance, the subjective sensation of throbbing is often most harassing, the heart action increased, violent, often tumultous, and its area of visible pulsation greatly extended.

May for a time exist alone.

Tremor.—A fine involuntary tremor usually attacks the head and extremities, and is of great importance, as constituting one of the earliest symptoms of the disease. *Tremor and rapid heart action may be present without exophthalmos or goitre.*

"Formes frustus" important.

Subsidiary and Complicating Symptoms.—A vast number of symptoms may be encountered in connection with the course of the disease. Ordinarily extreme nervousness, subjective sensations of throbbing, flashes of heat, excessive perspiration general or local, cardiac distress and mental irritability are those most marked. Less common are the marked gastro-intestinal disturbances, complicating myxœdemas, angio-neurotic edema, pigmentation or leukoderma of the skin, and serious mental depression.* Melancholia has been the common form observed, though acute mania sometimes occurs. Emaciation is often extreme in advanced cases; on the other hand one frequently meets with those of the well nourished type. The disease is ordinarily chronic, lasting for several or many years, it is moreover

Nervous symptoms marked.

Mental disturbances.

*In one case coming under the author's observation an insanity developed with delusions of so unfortunate a nature as to involve wholly innocent parties in a serious scandal the later development of the case making all clear.

frequently curable or may disappear under medical treatment. The author has thrice seen an acute firm enlargement of the thyroid gland associated *with marked nervous symptoms, tremor and tachycardia*, which lasted but three or four days.* Swelling of the thyroid gland, whether inflammatory or non-inflammatory, suppurative or non-suppurative, is not unusual and enlargement of the thyroid is so common in young girls at the age of puberty and in young women at the time of their first pregnancy as hardly to deserve notice. The surgical conditions of the thyroid cannot be considered here, but it should be said that the accessory lingual thyroid may attain a large size and that its removal is said to have been followed by myxœdema.

ACROMEGALY.—(Acromegalia. Marie's disease). **Definition.**—A chronic nutritional disease, characterized by progressive enlargement or over-growth of certain portions of the body, chiefly and primarily affecting the bones of the hands, feet and face, essentially chronic and progressive in its course and tending to a fatal issue.

Historical Note.—It was first described by Marie in 1886, having existed for thousands of years without recognition as a clinical entity, though presenting one of the most striking pictures known to medicine.

Etiology.—The cause of the disease is unknown, though the fact that in nearly every case that has come to autopsy, the pituitary gland has shown definite changes points to that curious structure as the probable source of the disease. The pituitary gland is a secreting organ as regards its anterior portion, whereas its posterior lobe has evidently a nervous function as its duct has become atrophied in the process of structural development, the secretion has become an internal secretion taken up by lymphatic absorption. There is much to support the belief that it is the centre for body growth and that it bears some curious but little understood relation to the thyroid gland. A relation between gigantism and acromegaly has been suggested and is probable. The disease occurs more frequently in women than in men, usually begins in early adult life, almost never after the age of 40, and no recognized pre-existing disease has been definitely connected with it.

Pituitary gland affected.

Symptoms.—It is essentially a case for street car diagnosis, being easily recognized by its outward signs. The face is elliptical in form, the superciliary ridges are pronounced, the head often large and massive, the lower jaw prognathic, the features enlarged, coarse by reason of the marked thickening of the greasy integument, in which appear deep creases, especially marked across the forehead. In many cases, even

Usually unmistakable.

*The last case two weeks.

the lids are thick and coarse, the hands and feet are enormously enlarged, the fingers are spatulate and clumsy, and upon examination the increase is found to depend upon the bony over-growth a finding easily verified by the X-Ray. This growth may extend for considerable distance upwards along the bones of the forearm and leg, and sooner or later tends to involve the clavicles, scapulæ, ribs and spinal column at which time the patient is likely to assume a peculiar stoop, due to kyphosis of the spine. There are no constant symptoms so far as the internal organs are concerned, and decided subjective symptoms, such as headache, mental irritability, malaise, joint pains, disturbance of vision

Fig. 161.—Acromegaly showing enlarged and clumsy bones (case of L. G.).

or drowsiness, may be absent until the later periods of the disease. The tendency is towards a fatal termination after a long term of years, in some cases covering two decades, and the symptoms are subject to many variations and exceptions. In a case which has been under the author's observation for nearly a decade, most interesting variations have been observed.* In this case the predominant facial hypertrophy

*Transactions of the Association of American Physicians, 1905.

in the bones of the face appeared in the upper jaw; the bones of the feet were but slightly hypertrophied as compared with those of the hand, yet after a term of years marked localized over-growth appeared in both

Fig. 162.—Acromegaly, showing predominant hypertrophy of great toe (case of L. G.).

great toes. Another rare phenomenon was a co-existing myxœdema, which promptly disappeared each time that a course of thyroid medication was applied, finally disappearing altogether at a time when a previously existing marked *enlargement* of the thyroid gland slowly

An unusual case.

subsided. In this case also there was from the beginning a chronic synovitis of the knee joints, persisting and so weakening the ligaments as to permit displacement of the patella and render the patient liable to dangerous falls. At the present writing this individual still lives, but grows gradually weaker and is subject to attacks of vertigo and occasional syncope.

Differential Diagnosis.—Acromegaly may be associated with gigantism and it is said may be mistaken for myxœdema, leontiasis ossea, osteitis deformans, arthritis deformans, and pulmonary hypertrophic osteoarthropathy. There is hardly a shadow of an excuse for any error along these lines. *Gigantism* need not be considered as the overgrowth is symmetrical. *Osteitis deformans* is rather a matter of deformity than over-growth, and in it the cranial bones are chiefly affected, rather than the facial bones as in acromegaly. Furthermore, the shape of the head in osteitis deformans is characteristic, its broadened cranial portions contrasted with the narrower maxillary region, a condition usually reversed in acromegaly. *Leontiasis ossea* shows merely bony tumors on the skull and face, and lacks every characteristic of acromegaly. In *pulmonary hypertrophic osteoarthropathy* enlargement of the hands and feet exists, but is confined chiefly to the articulations. The face is not affected, and chronic pulmonary disease of some sort is an invariable accompaniment.

OSTEITIS DEFORMANS.—(Paget's disease). **Definition.**—A rare disease characterized by kyphosis of the upper spine. A broad based thorax, lozenge shaped abdomen. Marked enlargement of the cranial portion of the head and enlargement and deformity of the long bones, due to a rarefying osteitis. The disease is extremely rare and of unknown etiology and needs no further description.

LEONTIASIS OSSEA.—A disease characterized by hyperosteitis of the bones of the cranium, rarely those of the face, in some instances combined with localized hypertrophy of the soft tissues.

MICROMEGALY.—This disease is the opposite of acromegaly, is excessively rare, and of unknown causation.

PULMONARY HYPERTROPHIC OSTEOARTHROPATHY.—(Bamberger's disease). This ailment, associated almost invariably with chronic pulmonary disease, is characterized by an enlargement of the hands and feet, the distal portions of the long bones, joints and terminal phalanges chiefly being affected. The finger nails become brittle and show longitudinal striation.

RICKETS.—An infantile disease characterized by general impair

ment of nutrition and peculiar changes in the bones. **Etiology.**—Poor food and unsanitary environment quite frequently associated with a syphilitic or tuberculous hereditary taint and relatively rare in breast fed children.

Symptoms.—The child appears delicate superficially and structurally with small bones, less rigid than normal and *showing at the epiphyses*, especially of the wrists and ankles, *characteristic swellings* found at autopsy to be associated with imperfect ossification. *In the parieto-occipital region the bone yields to finger pressure and in certain areas there may be a parchment like crackling.* The *broad forehead carries prominent frontal bosses* due to hyperostosis, *the ribs show the peculiar and characteristic beading* at the chondro-costal junction and there is frequently *pigeon breast* (*pectus carinatus*). The *abdomen is prominent*, the *liver and spleen are usually enlarged* and autopsy may show changes in the mesenteric glands. Such a child shows either an inability to walk or creep about or if walking may suddenly develop apparent weakness or disinclination. There may be slight fever, the *child is tender when touched or moved* and *extremely restless at night*, the pillow being soaked with perspiration and the hair often rubbed away at the occiput. As might be expected gross rickety deformities are common. *Spinal curvature, usually* but not always, antero-posterior appears and bowlegs of the most extreme type may be encountered. *Persistence of the fontanelles* is a common symptom. The diagnosis is not likely to be missed in well marked cases but the slighter varieties occurring under unexpected conditions may cause trouble.

Joints.

Cranio-tabes and frontal bosses.

"Ricketty rosary" and pigeon breast.

Backwardness and tenderness.

Persistence of fontanelles.

Fig. 163.—Hand:—Case of pulmonary osteo-arthropathy. (Preceding page.)

Prognosis.—The disease may indirectly cause a large mortality by weakening the child's resistance to other diseases but there is no direct

mortality. The development may be long delayed and permanent lack of resisting power and various deformities be left behind.

INFANTILISM.—Closely related to myxœdema and cretinism is the curious condition termed infantilism of the Lorain type or those described by Gilford as ateliosis and progeria. The former type (Lorain) represents merely man or woman in miniature. In the latter (Gilford) there is an asexual type representing delayed development and a sexual form represented by the ordinary traveling showman's dwarf, the delayed development in this case yielding along sexual lines at puberty. Progeria covers cases of infantilism associated with premature senility outward and structural. **Pancreatic Infantilism.**—This condition characterized by a marked amelioration under the administration of pancreatic extract is described by Byrom Bramwell.* In this case the developmental arrest was complete for 9 years but in the course of 9 months' treatment there was a gain of 8½ lbs. in weight and nearly 2 inches in growth. The patient was 18 years of age, his developmental arrest occurring at 11 or 12. The various classifications of infantilism are unsatisfactory and probably do not rest upon a very substantial basis.

OBESITY.—Obesity may be general or local, and its cause may be excessive food consumption, defective oxidation or elimination, lack of exercise or a distinctly hereditary tendency. In view of the discomfort caused by the excessive accumulation of fat, its tendency to limit proper exercise and impairment of the functions of vital organs such as the heart, one may fairly consider the condition as a morbid state, tending directly to shorten life. The general conformation of the body should be given weight and the large bones and unusual muscular development and a relatively small waist measurement are factors that materially modify the estimation of individual longevity. This means that *excessive fat* is the condition most feared.

Shortens life.

Lines to be drawn.

There are two distinct types of the obese, namely, the anæmic and the plethoric, with little to choose between them, so far as life expectancy is concerned. The obese are peculiarly liable to disturbances of the secretory organs, heart disease, asthma, diabetes, gall stones, gout, apoplexy. Furthermore, they succumb readily to severe acute infections, and are bad subjects for major operations.

General Comment.—As is well known, certain races or tribes deliberately cultivate obesity in their women, making the pudgy outlines of the Hottentot Venus their ideal of feminine beauty. In all races,

* Clinical Studies, Vol. I, part 2. Jan., 1903.

90% of the cases of excessive obesity occur in women, furthermore, the condition is often congenital. Cases are reported in which a baby 13 months old weighed 75 lbs., a child 4 years old weighed 256 lbs. In some of these less excessive cases the excessive fat of childhood disappeared largely in adult years. Cases of excessive weight have been reported that tax one's credulity to the utmost. Daniel Lambert is said to have weighed 730 lbs. A case was reported in Baltimore in which the weight was 850 lbs. and one from North Carolina is said to have reached 1000 lbs. These cases are ordinarily shortlived, and the fact that few really fat people attain advanced age cannot fail to strike even the lay observer. In a curious disease known as "adiposis dolorosa" the excessive accumulation of fat is associated with headache, pain, tenderness of tissues and mental disturbances. It would seem that these cases were probably *in many instances* actually examples of myxœdema, though it cannot be denied that sufficient evidence is at hand from reliable observers to establish such a disease as a clinical entity. In the true disease, the accumulation of fat ordinarily occurs in the form of bunches or nodules, though later becoming generally distributed. Its cause is probably trophic disturbance, and degeneration of the ultimate nerve filaments has been reported.

Adiposis dolorosa.

THE INFECTIOUS DISEASES.

TYPHOID FEVER.—(Enteric fever, typhus abdominalis). **Definition.**—This acute general infection is caused by the typhoid bacillus and characterized by continued fever, enlargement of the spleen, a peculiar exanthem, diarrhœa or constipation, tympanites, abdominal tenderness, the diazo-reaction in the urine, and the agglutination reaction of Widal.

Etiology.—The bacillus typhosus of Eberth (1880) is a small motile rod, possessing numerous flagella, easily grown in pure culture and staining readily though not by Gram's method. *Its development in the body results in the formation of agglutinins and precipitins responsible for the almost pathognomonic test of Widal.** Outside the body it resists cold and moderate dry heat, yet fortunately *direct sunlight destroys it in a few hours* and it is readily killed by ordinary antiseptics. *In water it lives but a few days if saprophytic organisms are present and in ice rarely longer than ten days or two weeks*, though instances have been

Eberth's bacillus.

Vitality of germ.

*The test also proved the germ which, though previously accepted by reason of its constant presence and the fact that it could be recovered and grown in pure culture, had not fulfilled Koch's laws.

reported in which living germs were recovered from ice after a period of nearly five months. *In superficial soil and in the fæces they may live for months unless exposed to direct sunlight or other unfavorable conditions, and this applies to dust and filter sand as well as ordinary earth.* In milk, sweet or sour, in butter or in cheese they may persist for months, indeed milk is a favorable culture medium. Infection arises ordinarily through germs cast off in the fæces or urine, and, indirectly conveyed to the water or food supply of the individual. Though possible, direct contagion under ordinary precautions is so rare as to be almost negligible.* It is evident that the germs may be conveyed by food, clothing, soiled fingers, house flies and even by dust, but almost invariably water or milk is the vehicle. One polluted spring may poison a whole city, and many American municipalities are criminally lax in the supervision of their water supply. Milkmen using water from infected wells for cleansing utensils or less innocent purposes frequently convey contagion to their patrons. Salads may become sources of infection when their components are grown on soil fertilized by infected material, and even oysters bedded along lines of sewage flow may become active sources of infection.

Modes of conveyance.

Distribution of the Germs in the Body.—The infection is a general one, and almost any body fluid, secretion or excretion, may contain them. The urine is a peculiarly dangerous source of infection, containing germs in at least one-third of all cases, and oftentimes for long periods after apparent recovery. The organisms are constantly present in the fæces during the active stages of the disease, though often they cannot be demonstrated by present cultural methods. Spleen cultures and those from the blood and rose spots, yield positive results in the majority of instances. They *may* be found in the bile and even in sweat, sputum, serous exudates or the foci of suppuration.

The urine a menace as well as fæces.

Modes of Entrance.—It is probable that all infections occur through the intestinal tract and in the vast majority of cases it is the intestine and its associated structures that show the most marked lesions.

Varieties.—Following Osler, we may group cases as follows:—(1). *Typhoid fever with marked enteric lesions.* (2). *Those of slight enteric lesions.* (3). *Cases with no discoverable intestinal lesions.* (4). *Mixed infection.* (5). *Pseudotyphoid cases* or those due to the so-called paratyphoid germs. The first group comprises the vast majority of all cases. Groups two and three represent extremely rare find-

* Osler reports about 20 cases of apparently direct infection as occurring during 12 years' service in Johns Hopkins Hospital.

ings. Group four should be confined to those cases in which the secondary infection is caused by germs which favor the growth, and intensify the action of the original germ. Such are the ordinary pyogenic cocci, the pneumococci and the colon bacilli. The fifth group comprises those cases denominated *paratyphoid.* In regard to these it may be said that they are due to the so-called paratyphoid bacillus of Achard and Bensaud (1896), *and the disease cannot be differentiated by its clinical manifestations,* which are identical with those of true typhoid fever. The absence of the Widal reaction with the Eberth bacillus in a case clearly typhoidal in type is the best diagnostic evidence if paratyphoid cultures are unobtainable.*

Pseudo-typhoid.

Pathologic Anatomy.—The germ may be recovered from the blood, mesenteric glands, spleen, bone marrow and intestinal lymph structures and readily cultivated. The almost constant and essential lesions are those found in the agminate glands known as Peyer's patches, and the solitary follicles of Lieberkühn in the ileum and jejunum, the process being one of medullary infiltration. The simple follicles of Lieberkühn are found in both the large and small intestine, Peyer's patches are found throughout the ileum and the lower jejunum. The follicles are at first grayish white and prominent, the inflammation increases during a week or ten days, then terminating by fatty degeneration and absorption, or more often in necrosis and ulcer formation involving the follicles alone, or exposing the submucosa and muscularis at the base of *oval* round ulcers, which unlike those of tuberculosis, tend to parallel the long axis of the bowel and lie opposite the mesenteric attachment, resulting in deep and extensive sloughing, the neighborhood of the ileocæcal valve being usually the point of maximum change. The ulcer border may be undermined, the edge regular or irregular and the stage of actual necrosis ordinarily represents the third and fourth weeks of the disease. Hemorrhages, trivial or serious, may occur at any time, from the beginning of the ulcerative process to the completion of that of healing but death *after cicatrization* is a rare event. Perforation with septic peritonitis occurs in about 5% of all cases and the danger of excessive tympanites during the later weeks of typhoid is evident. The perforation is ordinarily found in the ileum, rarely in the cæcum or adjacent portion of the colon, or vermiform appendix. *The spleen* is enlarged and extremely soft, rupture may rarely occur either with or without traumatism, abscess

Typhoid vs. Tuberculous ulcers.

* Furthermore the paratyphoid culture of one epidemic may not show constant clumping in the disease of other epidemics.

is rare, infarction not unusual. *The liver* is hyperæmic and shows signs of parenchymatous degeneration, rarely abscess, and the *gall bladder* not infrequently *contains the germs* and may be the seat of a cholecystitis. *Kidneys.* An acute nephritis or suppuration is rare, cloudy swelling and granular degeneration more frequent. *Heart Lesions.* Endocarditis and pericarditis are extremely rare. Granular and fatty degeneration of the myocardium is common but is seldom a cause of death. *The Arteries and Veins.* Emboli and thrombi are not uncommon, venous thrombosis being the rule and having its most frequent seat in the femoral veins. Such a thrombosis constitutes one of the complications of later typhoid. *Respiratory Organs.* Splenization and hypostatic congestion are extremely common, infarction occurs in about 5% of all cases but abscess, gangrene and pleurisy are extremely rare.* *Nervous System.* A specific meningitis in typhoid is extremely rare, severe headaches and delirium common. *Mesenteric Glands.* Their marked involvement is constant.

Milk leg.

Symptoms and Diagnosis.—No disease is *at times* more difficult to diagnosticate in its earliest stages, none save influenza and syphilis more interesting and protean in its forms, none more difficult to forecast and few more exacting in their demands upon the therapeutic resources of the clinician. Many cases are remarkably clear in their development and run an uneventful course, but the practitioner must recognize the *atypical* and beware of hasty conclusions in those apparently straightforward. The work of the last decade has greatly simplified the diagnosis of typhoid for those who have at hand the aid of the clinical laboratory or the simple means to be described, and clinical and microscopic tests have added enormously to our positive knowledge of the character and frequency of variations in type.

Protean.

Major Symptoms.—(a). *An insidious onset.* (b). *A continuous and somewhat characteristic temperature.* (c). *Enlargement of the spleen.* (d). *Rose spots.* (e). *A lowered pulse temperature ratio.* (f). *A low leucocyte count.* (g). *Ehrlich's diazo-reaction.* (h). *Agglutination test of Widal.* (i). *The recovery of typhoid bacilli from the stools, blood or urine.*

Minor Symptoms.—Of the minor symptoms, one may mention *nose bleed, gurgling and tenderness in the right iliac fossa,* the so-called *typhoid tongue* with its V-shaped red tip and brilliant edges, the *pea soup stools,* becoming a *brilliant ochre* in the later stages, *headache, delirium* and *profound prostration.*

* Ulceration of the larynx has been reported but has never been observed by the author.

Complications.—The two most important symptom groups are, those of hemorrhage from the bowel, and of perforation of the intestines.

The Typical Case.—(Rarely seen). There is first a period of *general malaise* (lassitude, aching head and limbs, loss of appetite, etc.), during which (or later) *nose bleed* may occur with diarrhœa or constipation, more generally the latter. This condition progressively increases, slight *fever* is apparent and the patient takes to his bed. *The fever rises each night higher than the night preceding, receding each morning about one degree below the temperature of the preceding evening.* The *face becomes flushed*, the *tongue* is *heavily coated, save the tips and margins*, which remain a bright angry red. An antecedent *diarrhœa* may now increase *or* replace *constipation*, or the latter condition may persist. *Mental hebetude* is marked and the *pulse*, heretofore somewhat slow and soft, *becomes weaker and tends towards dicrotism.* At the end of a *week or ten days pale red papules*, easily blanched by pressure, appear over the abdomen and lower chest usually in small numbers, and the *spleen may be readily palpable. Tympanites* may be a prominent and distressing symptom. By the end of the second week the fastigium is reached and all symptoms are intensified. Headaches and hebetude are replaced by a *low muttering delirium*, and the *pulse* grows more *rapid and dicrotic.* The *heart sounds are weaker*, there is a tendency to *congestion of the lung bases*, the *tongue grows dry* and hard and sordes tend to accumulate upon the patient's lips and gums, the *attitude of* the patient indicates *profound weakness*, he sinks down in bed, lies constantly in one position, which must frequently be changed by the efforts of the nurses or ward attendants. If diarrhœa is present, the *pea soup stool* of the earlier period *is replaced by the light ochre stool* of the latter typhoid. *Tympanites* is likely to be *excessive* and increases the danger of perforation. *During two weeks the temperature is practically continuous*, all symptoms are intensified, hemorrhage or perforation may occur and tendency to hypostatic pneumonia increases. In the *fourth week there is a recession of symptoms*, fever gradually subsides, the mind clears, the spleen shrinks under the ribs, tympanites lessens and the heart sounds and pulse are of better quality. Such a case offers no difficulty in diagnosis, but one must consider the common variations.

Insidious onset.

Rising fever.

Gastro-intestinal signs.

Exanthem.

Mental state.

Decubitus.

Period of General Malaise.—Our experience during the Spanish-American war has shown how *frequently there may be a sudden onset with chill and high fever.*

"Abortive" cases.

Nose Bleed.—Frequent but neither constant nor limited to typhoid

fever. *Gurgling and Tenderness in the Right Iliac Fossa.* This much overrated symptom may be found in any diarrhœal condition, and any *extreme early tenderness is more suggestive of an appendicitis than of typhoid fever*, moreover, in many cases of typhoid these symptoms are altogether lacking. **The Typhoid Tongue.**—Such a tongue is strongly suggestive of typhoid but is not always present, many may occur in other conditions associated with high temperature and gastro-intestinal disturbances.

Often atypical.

Fever.—"Step-ladder" temperature is somewhat rare *in its typical form*, and the use of cold baths has robbed it of much of its symmetry; moreover, as before stated, fever may occasionally rise rapidly with or without an initial chill, instead of gradually as in a typical case and terminate either by crisis or by lysis in a few days or after several weeks.*

Afebrile typhoid.

The So-called Abortive Typhoid.—The *abortive typhoids are much more common than was formerly supposed*, and still more extraordinary are those extremely rare cases lacking temperature, which have been verified by the most modern and exacting clinical tests.

The exception.

Diarrhœa.—This symptom is present only in a *minority* of the cases on milk diet, regular movements or constipation being the rule.†

Present in two-thirds.

Rose Spots.—These, when present, are valuable diagnostic aids and a great source of comfort to the practitioner groping for a clew. They are present in about two-thirds of all cases, ordinarily few, scattered and limited to the chest and abdomen, they may be profuse and widely distributed, but unfortunately, are not limited to typhoid.‡

Often obscured.

Enlargement of the Spleen.—In association with the general symptoms of typhoid this sign is of great value, but often an existing tympanites makes its determination impossible, and, moreover, it may be found in paratyphoid, acute miliary tuberculosis, malaria, septic-æmia and some of the tropical fevers.

Diagnosis and prognosis.

Disturbed Pulse Temperature Ratio.—A low pulse rate associated with high fever is a valuable confirmatory sign of typhoid infection, though by no means limited to this disease. *It is, furthermore, a valuable prognostic sign* indicating light infection or good resisting power. It should be remembered that pneumonia offers the same symptom,

*Such cases were frequent amongst the young soldiers in the last war.

†Speaking from personal observation.

‡A case coming under the author's observation a short time ago presented the typical rose spots, but failed to react to the Widal test, and proved to be a case of miliary tuberculosis.

and in either disease one may be misled in elderly persons, in whom a slow pulse may be the result of an obstructive aortic lesion or arteriosclerosis.

Recovery of the Bacillus from the Stools, Blood or Urine.—The introduction of the Widal test has made this difficult and unsatisfactory cultural method of investigation of little use to the practitioner. *Spleen puncture* will often yield a pure culture, but is neither necessary nor justifiable save in the rarest instances.

Seldom necessary.

Nervous Symptoms.—*These are essentially those of the "typhoid state,"* and *may* be seen in any disease associated with *overwhelming toxæmia.* The low muttering delirium of the typical typhoid, and less often the mental hebetude (torpor) may be entirely absent, or, in rare instances, replaced by an acute maniacal delirium, which constitutes a most serious complication.*

Mental state.

Misleading Variants.—*Of the many curious variations* in the general form of this disease may be mentioned (a) that associated with *profuse and exhausting sweats* which strongly suggest septicæmia, pyæmia, certain forms of malaria or ulcerative endocarditis; (b) those in *which the onset is so distinctly pulmonary* in its type as to lead to a diagnosis *of pneumonia* without regard to the underlying infection; (c) *the tonsillo-typhoid form, often accompanied by erythema and simulating scarlatina;* (d) the *renal form* in which symptoms of nephritis are prominent and misleading, and (e) the many variations due to complicating *abscess and necrosis.*

Sudorous.

Septic.

Pulmonary.

Scarlatinal.

Renal.

The Important Factors in Accurate Diagnosis.—*The essential thing is a proper appreciation of the lesser value of individual symptoms, and the greater one of the grouped signs.* Aside from the Widal test, no *one* clinical symptom suffices for a diagnosis of typhoid, but *many combined* reduce error to a minimum and of those mentioned, nearly all are *important in combination* but of slight significance if isolated. Three symptoms have been reserved for separate discussion because of their great *individual* value. These are, first, *a low leucocyte count,* second, *Ehrlich's diazo-reaction,* third, *the agglutination test of Widal.* It has been amply proven that $\frac{9}{10}$ of all cases of typhoid, *unless complicated* by a secondary infection, yield a *subnormal* or *low normal leucocyte count,* which *tends to progressively decrease as the disease increases in severity.* The exceptions represent, chiefly, blood concentration from vomiting or diarrhœa, or cold baths. A count of 15,000 may be regarded

Value of clinical picture.

Valuable single symptoms.

*A considerable number of true typhoids pass through the whole course of their illness without any marked mental disturbance.

as the maximum thus produced, and, on the other hand, even in complications counts as low as 1000 have been reported. *Absence* of a high count in the relation to a suspected complication does not furnish absolute proof of its non-existence but the *presence* of a high count with no profuse diarrhœa or vomiting is good proof of one. A moderate secondary anæmia is practically invariable in all cases. The "coagulation time" of the blood is increased during the height of the fever (favors hemorrhage), and shortened during convalescence (invites thrombosis).

Ehrlich's Diazo-reaction.—This much maligned, misunderstood and faultily applied test is one of great value in the exclusion of typhoid, but cannot be used save with a thorough understanding of the conditions under which it appears, its limitations and the true typhoid reaction color.* The test recommended is that of Ehrlich as modified by C. E. Simon and the author. (See p. 349.)

Limitations of the Diazo-reaction.—It cannot be too emphatically stated that *the presence of a true typhoid diazo-reaction† does not prove the disease to be typhoid;* with equal emphasis it may be said that *absence of reaction in a supposed case, provided the test be made at a proper stage of the disease, amounts to almost positive proof that typhoid is not present.* This statement iterated and reiterated by the author ten years ago, has, in the last year or two, been abundantly confirmed by other observers using it in connection with the agglutination test. *It may appear as early as the fourth day of a typhoid and is almost invariably present by the end of the first week or ten days. It becomes intensified as the disease progresses but rapidly fades as soon as the acme of injection is passed.* This latter fact is an extremely important one, and a failure to recognize it has led to many errors. The reaction is present in many of the *acute exanthematous* diseases, but if made with the higher dilution as described on p. 349, will be found absent in *malaria, most cases of appendicitis, pneumonia,* and the *earlier stages of acute miliary tuberculosis.* Indeed its late appearance in the last disease is of considerable diagnostic value in that connection.

Value in negation.

Period of persistence.

Diseases excluded.

The Agglutination Test of Widal.—*This is by far the most important and positive test of typhoid fever.* It was first announced to

*"The Diagnostic Value of Ehrlich's Diazo-reaction," Greene, Journal of the American Medical Association, Feb. 24, 1894. "Recent Aids in the Differential Diagnosis of Typhoid Fever," Greene, Medical Record, Nov. 14, 1896.

†Viz.:—that diazo-reaction which is so intense as to yield a brilliant crimson band and upon shaking a pink foam.

the medical profession by Widal in 1896.* This test really depended upon the earlier discovery by Pfeiffer (May, 1894), who found that cholera vibrios mixed with the serum of an immune animal and injected into the peritoneal cavity of a guinea pig lost their motility and changed their form, and later on found that the typhoid germ reacted in a like manner with the blood serum of typhoid patients and applied the test to the identification of the germ. Widal *reversed* the process and applied it to the diagnosis of the disease. *The test is as nearly pathognomonic as any in the range of medicine, and in its simplest form may be applied by any practising physician.*† The method originally employed by the author, though rough and simple, was definite in its results and required merely an agar-agar (i.e. solid) culture of typhoid bacilli, a medicine dropper, a platinum loop, and distilled water. The loop being made of fine wire was so constructed as to leave a central opening of not more than one-sixteenth of an inch. The tests were made either with the fresh blood or the dry film, the latter being treated with a drop of distilled water before testing. Instead of the "hanging-drop" method, the ordinary flat preparation was used with equally good results. It was only necessary to place a drop of distilled water on two clean microscopic slides, take a small particle of the culture with a sterilized platinum loop and stir it into the drop of water. Sterilize the loop, and from a drop of blood obtained from the suspect allow the tiny loop to fill and stir this film into one of the drops containing the bacilli. A cover-glass was then dropped upon it, it was placed under the microscope and a reaction was indicated by loss of motility, and grouping of the germ, *half an hour* being the time set for a positive reaction, and reference being made to the control slide for error due

Practically pathognomonic.

Rapid rough method.

*Bulletin Medicale, 1896, page 618. The papers of Wyatt Johnston (New York Medical Journal, Oct. 31, 1896) and of the author (Medical Record, Nov. 14, 1896, and Dec. 6, 1896), being the first reports published in this country.

†At the present time one may obtain the so-called typhoid agglutometer (Parke, Davis & Co.) by means of which the test may be applied without the use of the microscope. The agglutometer comprises three tubes containing (a). A sterile permanent suspension of typhoid bacilli. (b). A control tube of the same nature for the comparison of reactions. (c). A dilution tube containing fluid for properly diluting the serum previous to testing. (d). Tube for the collection of the blood to be investigated. (e). A pipette for the withdrawal of the serum. (f). A puncture needle. The test is made by adding two drops of the serum to the fluid in the diluting tube and this diluted serum is distributed in specific quantities in three tubes of suspended material. If the reaction is positive floccules appear in one or more of the tubes in one or two hours, the reaction being readily determined by a comparison of the treated tubes with the control.

Many methods.

to pseudo-reactions. Many methods are now in use providing for exact dilutions and the use of bouillon cultures, some far too complicated for the practitioner and requiring laboratory facilities, but from actual experience the author can recommend the extremely simple one above mentioned.* During this early period in tests thus made it was found that *if a relatively large amount of the culture material was used a positive reaction could be determined in the same time and by the same method, without a microscope, through the appearance of a dust-like film,* which became visible when the surface of the cover-slip was viewed tangentially against the light. It may be added that in any event, oil immersion lenses are not absolutely necessary to the performance of the test, lenses of moderately high power being quite sufficient. The author firmly believes that the simpler methods add rather than detract from the accuracy of this valuable test and that a solid culture and relatively low dilution 1–20 give the best results. *Slides and cover-glasses should be washed in pure water before using to remove any powerful antiseptics. Blood should never be taken upon paper or cardboard, but rather upon glass, porcelain or tinfoil.*

Author's macroscopic test.

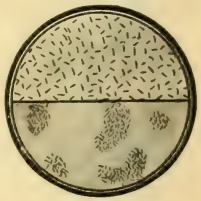


Fig. 164.—Typhoid agglutination test. (Widal.) Upper segment shows the freely moving germs. The lower the typical "clumping."

Time of Appearance.—If the fresh blood be used the reaction may be found as early as the fourth day of the disease. It is persistent throughout the illness and in some cases for years afterwards, but its exact duration has not been determined. Cases of delayed or even absent reaction have been reported, but the author has found none which have been delayed beyond the first ten days of the illness. *A single negative test should never be considered sufficient.*

As early as fourth day.

Persistence period unknown.

Value of the Test.—Even working with dried blood, competent observers report positive results in at least 95% of the cases tested. It corrects innumerable errors of observation and must be given not only first place in the diagnosis of typhoid, but a value at least *equal* to that of all other symptoms combined. It should be universally applied in city and country practice alike, and its teachings accepted in a proper spirit of humility.

Its use imperative.

DIFFERENTIAL DIAGNOSIS.—Widal's reaction when practicable will almost invariably eliminate any doubt that arises, but in its absence one must depend upon the grouping of symptoms.

The Widal conclusive.

*"Recent Aids in the Differential Diagnosis of Typhoid Fever" (Medical Record, Nov. 14, 1896).

Acute Miliary Tuberculosis.—This differs from typhoid in its irregular temperature, the fever being often of the inverse type; chills and sweats, more rapid emaciation, the fact that rose spots are usually absent, the late appearance of the diazo-reaction and splenic tumor and sometimes in the discovery of definite pulmonary symptoms.* The family history or known exposure to infection is often suggestive, but it must be remembered that *typhoid and acute miliary tuberculosis may co-exist* and that *in both diseases there is a tendency to abnormally low leucocyte counts.*

Error seldom excusable.

Septicæmia.—As this disease is usually characterized by a distinctly "*typhoid state*," the *focus of suppuration must be sought*, and the irregular temperature, with possibly chills and sweats, and a marked leucocytosis may point to a true diagnosis. It gives the diazo-reaction exactly as does typhoid. *Ulcerative Endocarditis, Salpingitis.*—Present much the same features with the added help of *localized symptoms.*

May require Widal.

Appendicitis.—This constitutes a not infrequent source of error, operations for appendicitis being made without justification and cases of appendicitis being treated as typhoid. Ordinarily, the localized symptoms and sudden onset with perhaps a previous history of similiar attacks serve to make the diagnosis clear. *Early* localized tenderness in the appendiceal region is rare in typhoid and in appendicitis the early colicky pain is almost invariable and the Widal test is negative.

Symptoms usually distinctive.

Meningitis.—This is sometimes to be distinguished from the cerebral form of typhoid only by the absence of the diazo-reaction, the failure of the Widal test, and diminished urinary chlorides, but ordinarily, no confusion of the two diseases is possible.

Simple Continued Fever.—Can never be positively differentiated unless the two crucial tests are applied.

Pneumonia.—Is a frequent complication of typhoid, but as an independent disease may be recognized by its sudden onset, marked localizing symptoms, high leucocyte count, absence of true diazo-reaction in high dilution (1–100) and of the Widal test.

Malaria.—The remittent form can at times be differentiated only by finding the plasmodium in the blood, the absence of a diazo-reaction and a negative Widal test. In any malarial country a mistake in diagnosis is extremely frequent.

Clinical "*bete noir*."

Paratyphoid.—As before stated this disease exactly simulates typhoid and can only be differentiated by the agglutination test. It

*The author places much reliance upon general pulmonary *hyper-resonance* in the earlier stages.

Widal's test the only means.

unquestionably furnishes an explanation of the few cases in which a failure of the Widal test has been reported in the face of a group of symptoms convincingly typhoidal in type and such failures can scarcely be proven without cultural or paratyphoid agglutination tests.

Conclusion.—*It is evident that both in direct and differential diagnosis the agglutination test may be the only determining factor, and that no practitioner of the present day can justify himself for its neglect.*

Prognosis.—The mortality of typhoid varies greatly according to the age of the patient, his condition at the time of its onset and the character of the epidemic, if such exist. It varies in private practice from 5–15%, in hospital practice it may reach 20%. Much depends upon the character of the hospital population and methods of treatment employed. The mortality of different epidemics varies widely in the presence of virulent infection, a picked body of men such as would be represented by a newly mustered volunteer regiment furnishing a low mortality. On the other hand, the same regiment after a long campaign is likely to furnish an extreme mortality.* The female furnishes a higher mortality than the male, and the so-called walking typhoid, i.e., the ambulatory form, presents, as might be expected, an excessive death rate. *The temperature curve seems not to figure greatly, but pronounced nervous symptoms and high pulse rate, excessive meteorism and such complications as hemorrhage and perforation are of bad portent, the latter being almost invariably fatal* even if prompt surgical measures are instituted. Hemorrhage occurs in about 5%, and perforation in about 3% of the cases. *There is a marked tendency to relapse, especially if improper feeding be permitted during the period of convalescence and it is seldom wise to depart from the routine fever diet until the temperature has been normal for a week*, though it sometimes requires the finest judgment to decide whether it is due to an obscure complication or the persistence of the infection and intestinal changes. *The best guide is the condition of the abdomen; freedom from tenderness* and rigidity (general or localized) being the safest indication for an increased dietary.

Private vs. Public.

Epidemic variants.

Physical condition.

Sex.

Symptom values.

Relapse.

Increased dietary.

INFLUENZA.—(La grippe, la cocotte, la follette, the "pleasant acquaintance," the "jolly rant," the "knock me down fever," catarrhal fever, epidemic catarrhal fever.)

Its misnomers.

*This is well illustrated by a comparison between the death rates of such of our own volunteer regiments as remained in camp, but out of service during the Spanish-American war, and the British regiments in South Africa, amongst whom the death rate was nearly trebled.

Historic Note.—Influenza has been known and recognized as a clinical entity for over 2000 years, though its true cause remained obscure until 1892, when Pfeiffer discovered its specific micro-organism. Its peculiarities led to many curious theories of causation, and to such extraordinary names as appear above. Hippocrates ascribed it to the interposition of Providence, Sydenham to some "occult and inexplicable changes wrought in the bowels of the earth." Weber would hold to a "negative state of electricity," and recommended the wearing of socks lined with non-conductors. Fogs, comets, earthquakes and various other disturbances were supposed to be concerned in its causation and over two and a half centuries ago there were advocates of a "contagium vivum." Influenza appeared in the Greek army at the siege of Syracuse and destroyed thousands of soldiers. In 1782, the fleet of Admiral Kempenfeldt was obliged to put back to port on account of the destruction of his crews and in 1728 it drove the imperial court of Russia from the city of Moscow.

Early theories.

Ancient epidemics.

General Comment.—The enormous importance of influenza is hardly recognized even by the scientific world. Like cholera it follows lines of commerce and one can hardly escape the belief that it is always present in a large portion of the inhabited world. It would seem that *more should be done in the way of quarantine and prophylaxis*, for in spite of the many difficulties it offers, the author's experience would indicate that prompt isolation of an infected individual may prevent the infection of the remaining members of the household.

Etiology.—The influenza bacillus of Pfeiffer is readily obtained from the secretions or from the blood of those suffering from the disease. It is small, non-motile and readily stained with concentrated stains such as alkaline methylene blue and carbol-fuchsin, but so deeply at the ends as to resemble diplococci. The bacilli are Gram negative, are cultivated with difficulty on blood agar and are solitary or less often in chains. They seem to thrive in the mucous discharges and may not only infect others, but, after a considerable period may also reinfect the individual. *The disease does not confer immunity, but seems rather to render its victim more susceptible to other attacks.*

The germ.

One attack invites others.

Incubation.—The exact period is unknown, but varies from 24 hours to one week. Two or three days is probably the average duration.

Symptoms.—There are two classes of influenza, *first, true epidemic influenza, second, endemic-epidemic influenza*. The former is usually the more serious and fatal type, but aside from the severity of its

Chief types.

Protean.

symptoms and complications, is precisely like the second form. Moreover, while possessing certain fairly well defined peculiarities, it is the most protean of *acute* diseases. *Profound exhaustion*, mental and physical, is its most constant feature, the *mucous membrane* and particularly that of the air passages its favorite seat, *and, it attacks the nervous system with especial violence.* Moreover, the epidemics of different years assume certain predominating features, some are especially catarrhal, taking the form of coryza, bronchitis and pneumonia, in others, the nervous and cerebral types predominate, and in yet others the gastro-intestinal tract. Usually distinctly febrile, it may *present an afebrile form or one in which the fever is so slight as to resemble an ordinary cold.*

Coryza-like attacks.

These latter types are especially common in the endemic-epidemic forms. *Its diagnosis*, in the absence of a definite epidemic and even in its presence, *is oftentimes rendered obscure by its simulation of a multitude of diseases, resulting either from its selective points of attack or from complications.* Amongst these confusing conditions may be mentioned cerebral and cerebro-spinal meningitis,

Diseases simulated.

cerebritis with hemiplegia or paraplegia, intractable neuralgia, pleurisy, pneumonia, jaundice, vomiting, diarrhœa, dysentery, nephritis, suppurative or non-suppurative otitis media, endo- or pericarditis, thrombosis, embolism, melancholia, mania, neurasthenia, iritis, conjunctivitis and optic neuritis. In view of these complicating conditions as relating to diagnosis there is but one safe ground for the clinician to occupy, and that is *to recognize and act upon the fact that in the absence of an epidemic, influenza may sometimes be recognizable only through the recovery of the bacillus and the appearance of a degree of exhaustion entirely out of accord with the other symptoms presented.*

Cardinal points.

Mistakes in diagnosis must inevitably be common, excusable, and fortunately in most instances without serious results, inasmuch as the treatment of most complications is essentially that of the disease which they simulate.

Common type.

In by far the greater number of cases the symptoms are primarily those of an acute coryza and bronchitis. The pneumonias of influenza are commonly broncho-pneumonic, though lobar pneumonia is very common,

Influenzal pneumonias.

and either form may be due to an infection in which the bacillus of Pfeiffer directly participates. The author has observed two examples of pneumonia occurring in connection with influenza, both apical, with a marked and typical pneumonic onset, and characteristic physical signs associated with nausea, vomiting and diarrhœa, and rapidly assuming a dysenteric form. The cough, though urgent, was unproductive, there was no localized chest pain *and in 24 hours all physical*

signs had disappeared, the fever subsided by lysis, the dysentery disappeared with the subsidence of the physical signs in the chest, *but in each case the exhaustion was extreme and the recovery of strength unusually slow.* These cases are quoted merely to illustrate the extraordinary departures from the type to be seen in every influenza epidemic. A complete consideration of the vagaries of the disease would demand a large volume.

Illustrative cases.

Prognosis.—As has been said, *influenza is a disease of enormous actual but low relative mortality,* that is to say that considering the universality of an epidemic and the enormous numbers of individuals attacked, the death ratio is small, yet the total number of deaths is enormous and the indirect mortality adds greatly to the figure. Every practising physician knows how frequently one attack of influenza has proven to be the starting point of others and how often it has been the last straw in the case of those of failing health, whether from pre-existing chronic disease or old age. In the year 1900, over 16,000 deaths were reported as due to influenza and it is probable that if one could get at the facts as to the indirect effects of the disease, such mortality figures would be multiplied fivefold. This enormous mortality, the fact that it kills the adult by preference, and especially those engaged in active productive work, should open the eyes of anitary authorities to the necessity for quarantine regulations, both as applied to the State and tto the household.

Actual vs. Relative mortality.

Potential wealth loss.

Fig. 165.—Cholera Bacillus (comma bacillus or spirillum choleræ of Koch). Non-sporogenous, flagellate, motile, parasitic, saprophytic, non-chromogenic aërobic and facultative anaërobic, liquefying, spirillum, readily stained but Gram negative.

ASIATIC CHOLERA.—("The death blow").

Definition.—*An acute injection caused by the comma bacillus of Koch and characterized by profuse rice-water diarrhœa, violent muscle cramps and collapse.*

Historic Note.—It is one of the most ancient of diseases in the East, but of comparatively recent development in the United States, the first epidemic having occurred in 1832.

Etiology.—Koch reported his discovery of the comma bacillus in 1884. The germ retains its vitality for a week or more on food stuffs such as butter, milk and meat, lives but a day or two in oysters, but finds sewage a good culture medium. Water is a great conveyer and flies also carry it, but it travels no faster than man, following the trade channels, whether these be railways, canals, rivers or steamship

lines, tending to spread from India, where it is endemic, to all parts of the civilized world. The great pilgrimages, fairs, and festivals serve to disseminate it widely; filth, over-crowding and infected water and food being part and parcel of such gatherings.* The disease is readily conveyed by fomites but is essentially water borne. The city of Hamburg in 1892–3 had 18,000 cases with a mortality of over 40%. Altona, a suburb, had but 516 cases, both cities drank the same water, *but Altona filtered hers.* One convalescent workman from Marseilles once infected the water of a large reservoir, and through it a large district of the city of Paris. Cholera is a hot weather and low altitude disease, the germ is readily destroyed by sunlight, but vast quantities of bacteria are discharged in the stools and many objects may serve as indirect carriers. It is not highly contagious and scrupulous care in regard to the dejecta permits nurses and physicians to perform their duties without fear of infection; indeed fear undoubtedly favors infection. Age and sex seem to cut no figure as regards disease incidence, and neither absolute nor relative immunity is well proven.

Morbid Anatomy.—Rigor mortis is extraordinarily rapid, producing horrible post mortem movements of the extremities, lower jaw and even of the eyes. The tissues are dry and shrunken, the blood is thick and tarry and there is an intense congestion of the stomach and intestines, and cloudy swelling and parenchymatous degeneration of the viscera.

Symptoms.—*The incubation period is from two to five days.*

Preliminary Stage.—This *lasts but a few hours and presents malaise, marked mental depression, headache, diarrhœa, nausea, vomiting and colicky pains.*

Stage of Purging and Collapse.—This is characterized by profuse *and almost constant serous discharges from the bowel,* accompanied by *tenesmus, intense thirst and excruciating muscle cramps* especially affecting the abdomen and calves of the legs. *Vomiting* is almost incessant, the *countenance is Hippocratic, cyanosed and shriveled,* the *skin is wrinkled, moist and cold,* the *superficial temperature is greatly subnormal, though the rectal temperature may reach* 103–105 F.

Typical attack.

The Blood.—There is both *polycythæmia* and *leucocytosis,* the red cells running from 6,000,000–8,000,000 and the leucocytes from 14,000–

*The British have found the utmost difficulty in checking its development and dissemination. Native visitors to Holy shrines bathe in Holy wells and drink of the precious water which contains the washings of many bodies. Sick and well are crowded together, dirt abounds, ventilation is deficient and in event of an epidemic the care of the sick and the proper disposition of the dead are alike neglected.

60,000 per c.cm. The *hæmoglobin percentage is high* and the blood is obtained with difficulty. The *pulse* soon becomes excessively *rapid, weak, flickering, or even absent at the wrist,* the *urine scanty or suppressed,* the *tongue and throat dry, yet the patient sweats profusely.* The *stools are of the* characteristic "*rice-water*" type and *the patient becomes comatose, or passes into the third stage.* The *duration of this stage* of collapse is from a few hours to two days.

Third Stage.—(*Reaction.*) All symptoms gradually subside and a tedious convalescence ensues Various late eruptions may occur.

Prognosis.—The mortality varies from 25–75% in different epidemics, death may occur in relapse, in cholera typhoid when the delirium is marked, and coma rapidly supervenes, or the patient may die even before the rice-water stools appear (Cholera Sicca). The so-called *cholerine* is a very mild form of the disease, accompanying certain epidemics and isolated cases offer great difficulty in diagnosis.

Diagnosis.—In the absence of an epidemic, cases of poisoning by certain minerals, ptomains or mushrooms may cause difficulty. Cholera nostras (cholera morbus) in its severer forms exactly simulates true cholera.

Positive Differential Symptoms.—(a). *The agglutination test,* which is essentially the same as that for typhoid fever, is said to be positive as early as the second day. (b). *The germ may be recovered and cultivated from the stools* and the diagnosis thus absolutely established.

BUBONIC PLAGUE.—("Black death," "pest," malignant adenitis). **Definition.**—A *virulent epidemic infection* caused by the *bacillus pestis* of Yersin, and characterized by *high fever, great prostration,* the *formation of buboes, a tendency to hemorrhage,* both subcutaneous and from mucous membranes, and *a remarkably high leucocyte count,* highly infectious but not markedly contagious.

Historic Data.—It is as old as the Pyramids. After the 6th century it repeatedly visited Europe and Great Britain, killing during less than one century in London alone, 161,344 people, indeed, the epidemic of 1665 caused 68,000 deaths, the total population of London being at that time under 500,000. The Indian plague of 1889 is said to have been responsible for at least 250,000 deaths and in the 14th century it is said to have carried off not less than one-fourth of the total population of Europe. It is endemic in China and India, and a frequent visitor to the Philippines. An ancient scourge.

Etiology.—It is distinctly a *filth disease,* requiring for its proper

development, dirt, over-crowding, poor food and a warm moist climate. The germ is an encapsulated, short, round micro-organism. Its habitat is the soil, and it may be conveyed by fomites, including food stuffs, such as butter, milk, cheese, green vegetables, by water or by sewage. It lives long and thrives in urine, sputum and fecal matter. Lice, fleas, flies and mosquitoes serve as carriers. Dogs, cats and rats become infected and serve as vehicles of contagion. These facts greatly increase the difficulty of stamping out an established epidemic.

Readily conveyed.

Difficult to control.

Fig. 166.—Bacillus of Bubonic plague (bacillus pestis of Yersin). Minute, non-motile, non-sporogenous, non-chromogenic, pleomorphous, aërobic and facultative anaërobic organism easily cultivated and stained but Gram negative.

Morbid Anatomy.—That of an acute infection with profound toxæmia. The lymph vessels are chiefly affected, the glands being enlarged, edematous and hemorrhagic, or showing suppuration and external ulceration. The bacilli are generally distributed, the kidneys and spleen are hyperæmic and the lungs may show pneumonia and infarction. Primary and secondary carbuncles, ecchymoses and dermatitis may be present and the viscera generally show fatty and parenchymatous degeneration with marked hyperæmia.

Profound toxæmia.

Pestes Major.—Symptoms.—Incubation period 2 to 10 days. Prodromal period from 24 to 48 hours. *Pallor, vertigo* and *profound muscular weakness are associated with nausea, vomiting and diarrhœa. Nose bleed* is frequently present and *mentaldepression marked.* The *fever rises steadily until just before the active stage.*

Active Stage.—This is characterized by *cthill, high fever* (104–106° F.) and *rapid pulse* (120–200). The *expression is anxious, countenance livid, conjunctivæ congested, the skin dry and hot.* After three or four days *buboes* appear in from 70–80% of the cases. Those of the groin are affected primarily in 60% of the cases. *Suppuration is a favorable sign, but gangrene may supervene.* Petechia or extensive ecchymoses may appear (plague spots), as may other skin eruptions of various types. *Any mucous membrane may be the seat of hemorrhage.*

Buboes.

Plague spots.

High counts.

The Blood.—The leucocytes average more than 90,000 to the c.cm. and there is marked polycythæmia, the average count of red cells being about 7,000,000 per c.cm.

Virulent types.

Varieties of Pestes Major.—(a). *Septicæmic plague.* In this form buboes do not appear, the organism is overwhelmed and death occurs in a few days or hours. (b). *Pneumonic plague.* In this the

lesions are chiefly and ordinarily those of broncho-pneumonia, the sputum is loaded with germs and about 90% of the cases die.

Pestes Minor.—This mild form has *slight fever of short duration, glandular swelling with or without* suppuration, and may easily be misdiagnosed. *Such cases are usually forerunners of epidemics.* Misleading form.

The Diagnosis.—Lacking an epidemic, the diagnosis must sometimes depend upon the recovery of the germ, as the agglutination reaction though often obtainable, is not absolute and occurs late in the disease. Reliance is to be placed chiefly upon (a). *Prevalence of an epidemic.* (b). *Profound exhaustion.* (c). *Anxious countenance and mental depression.* (d). *Buboes.* (e). *Petechiæ* (f). *Hemorrhage.* (g). *Leucocyte count.* Cardinal signs.

Pathognomonic Sign.—There is but one, viz.:—*the recovery of the bacillus from the blood, urine, sputum or feces.*

Differential Diagnosis.—*Tuberculous adenitis.* Fever absent or intermittent, development slow and *comparatively* painless; this could hardly be confounded even with pestes minor. *Syphilis* presents initial lesion or cicatrices, sore throat, characteristic eruption of mucous patches and its buboes do not suppurate. *Chancroid*, initial lesion, strict localization of bubo. *Hodgkin's disease* and *gonorrhœal bubo* could only be confounded with pestes minor by sheer stupidity or carelessess. Usually easy.

The mortality is always large and even in those inoculated with antiplague serum it is said to be 14%. In some epidemics it runs from 50–95%. Prognosis.

DENGUE.—(Deng-ga). ("Break bone" or "dandy" fever). **Definition.**—*A tropical and sub-tropical disease* of *low mortality and high infectivity, characterized by its sudden onset, severe muscular and joint pains, irregular rashes, double febrile paroxysm and tendency to relapse.*

Etiology.—It is probably due to an organism resembling the malarial plasmodium, reported by H. Graham, of Beyrout, Syria, in 1903. The *culex fatigans* or common mosquito of Beyrout is said to be the intermediary host and source of infection. A mosquito host.

Prognosis.—There is practically no mortality. *Incubation period.* Three to five days, no prodromata.

Symptoms.—The *onset is sudden* with *chill, high fever* and *rapid pulse, headache, backache, muscular, joint and deep seated bone pain associated with extreme tenderness.* Both large and small joints may be swollen. *Red and enlarged lymph glands* are frequent, *vomiting* may occur, the *face* is deeply congested *and a rash* of short duration. Arthritic onset.

irregularly distributed, and variable in character is often present. One of the *initial symptoms* may be *extreme pain in the eyes.** *On the second, third or fourth day a crisis occurs* with the usual phenomena, and is *followed by a period of remission* of all symptoms, *lasting from two to five days, then follows a reinvasion* less severe than the first, *terminating in a second crisis after three or four days or even a few hours, and during this stage* a somewhat characteristic rubeolar eruption may appear, first on the hands, then over the body, but always most markedly over the hands, wrists, elbows and knees, occasionally forming a diffuse red rash by *coalescence.* Desquamation follows and is usually furfuraceous. A second remission and third paroxysm may occur, or the first remission may terminate the disease. In more severe cases, jaundice, black vomit, marked albuminuria and hæmaturia may occur and suggest "yellow jack."

Remission.

Terminal eruption.

Severe forms.

Differential Diagnosis.—(See yellow fever.)

YELLOW FEVER.—("Yellow jack," "bronze john.") **Definition.**—A virulent infectious but non-contagious disease, characterized by sudden onset, high fever, jaundice, black vomit and slow pulse.

Etiology.—Though the germ is undetermined, a commission, headed by the late Walter Reed, U. S. A., in 1900–01 proved the *intermediary host and infectious agent* to be the mosquito known as the Stegomyia fasciata. The work of this commission furnished one of the most brilliant examples of heroic self-sacrifice and scientific acumen in the history of medicine. Dr. Lazear became infected and died of the disease, non-immune volunteers slept for 20 days in the stained sheets taken from the beds of yellow fever patients, their heads wrapped in soiled, bloody cloths, yet all these, being protected from mosquitoes, escaped the disease. Those isolated for long periods and then exposed to multiple inoculation by mosquitoes that had fed upon yellow fever patients, in nearly every instance developed the disease in its ordinary form. It was found that mosquitoes required 12 days to develop virulence, and retained their infectivity for 8 weeks or more. A subcutaneous injection of yellow fever blood produced the disease even though the blood were defibrinated and passed through a porcelain filter. *The experiments proved that the only quarantine measures necessary were those calculated to destroy mosquito life in houses or on shipboard, and that the disease is not conveyed by fomites; further, that to control an epidemic it is only necessary to screen the sick and the houses of the well, destroy all resident*

Major Reed's great work.

Heroism of volunteers.

Period of virulence.

Blood infective.

Simple preventive measures.

*This may also be marked in yellow fever.

mosquitoes and to treat marshes, stagnant pools, water barrels and other breeding places with petroleum which destroys the surface breathing larvæ. Through such measures, Santiago, Havana, and other towns were rendered free from yellow fever by the government medical authorities after the Spanish-American war. Substantial proof.

Seasonal Influences.—All that was previously written regarding these matters now resolves itself into a discussion of mosquito life and propagation and it has always been recognized as a summer and early autumn disease, killed by frost. **Race.**—The whites are chiefly affected, though negroes are not wholly immune and visitors contract the disease more readily than those acclimated. **Sex.**—There is equality of susceptibility, though on account of greater exposure males chiefly predominate. **Age.**—It resembles typhoid in its preference for the young adult. Old people and infants ordinarily escape, the latter probably from minimum exposure. **Immunity.**—One severe attack protects for life.

Morbid Anatomy.—Diffuse nephritis, gastric hyperæmia with hemorrhagic stomach contents, hemoglobinæmia, general glandular enlargement, cutaneous hemorrhages and deep jaundice are the usual findings.

Symptoms.—The first stage strikes "*like lightning from a clear sky,*" with *chill* and *high fever* (103–106 F.) accompanying a *singularly slow pulse* (80 to a temperature of 104 F.); there is *excruciating pain in the loin and in the leg muscles, profound muscular weakness, violent headache, sore throat and vomiting, and the mental depression is often marked.* A *slight jaundice commencing in the eyes and extending to the chest, arms and body may appear even in this stage.* "A bolt from the blue." Early jaundice.

Physiognomy.—The *expression* is thought by Guiteras to be quite characteristic. *The cheeks and conjunctivæ are congested, the eyes staring, humid and ferrety, the general expression "anxious." Albuminuria may be marked as early as the second or third day.* There is *photophobia* frequently associated with intense orbital headache, *vomiting,* increasing in intensity until black vomit may appear; the *tongue is swollen and beefy* or often small and pointed, *and the gums are tumid.* The *pulse is not only primarily slow, feeble and compressible but tends to decline even with rising temperature.* Characteristic facies. Black vomit. Slow pulse.

Second Stage.—(From a few hours to 36 hours.) On the third or fourth day all symptoms abate leaving the pulse slow (even 30 or 40) and compressible, the temperature may be normal and recovery follows in mild and favorable cases, though convalescence is often protracted and accompanied by irregular fever. Remission.

Third Stage.— In severe cases a stage of intensification of the initial symptoms and collapse follows a remission. Its duration is from one to three days. The temperature usually rises slowly to 102° or 104° F. or may rise steadily until death, though the pulse may fall to 60 or 01 even 30 beats per minute. The *jaundice deepens, black vomit occurs, albuminuria is invariable, hæmaturia commences, total urinary suppression is not unusual and hemorrhage may occur from the nose, gums, lips, rectum or any other mucous membrane, the skin* may be of a *deep mahogany color* and various irregular eruptions may occur. *The late Dr. West* of *Galveston always insisted that the renal symptoms overshadowed all others in severe cases.*

Falling pulse.

Hemorrhages.

Renal symptoms.

Prognosis.—It is largely determined by the height of the original fever. In Sternberg's analysis of 269 cases, of those characterized by an initial temperature of 105–106° F., 61% died; of 104 to 105° F., 30% died; of 103 to 104° F., 6% died; of 103° F., none died. *Recovery may ensue in any case however severe,* but convalescence is slow.

Relation to fever.

Diagnosis.—*Essential Data.* Mild cases may be unrecognizable, but in marked cases one especially notes:—(a). *Sudden onset without prodromata.* (b). *Presence of an epidemic or history of exposure.* (c). *Physiognomy.* (d). *Low initial pulse rate with high temperature.* (e). *Falling pulse rate and volume with rising temperature.* (f). *Excessive gastric irritability.* (g). *Early jaundice and later extreme pigmentation.* (h). *Black vomit, epistaxis and bleeding gums.* (i). *Albuminuria with a tendency to suppression* (predominance of renal symptoms). (j). *General tendency to hemorrhage.* (k). The slight *apparent* loss of red cells per c.mm. is a marked feature in view of the hemorrhagic conditions.

Differential Diagnosis.—*Dengue,* no mortality; joint symptoms usually more pronounced and *persistent,* yellow fever pulse-temperature ratio absent; jaundice seldom marked and rarely as *early* as the 3d day; black vomit usually absent, renal symptoms not so prominent or so early, rashes common and somewhat characteristic and hemorrhages rare save hæmaturia. *Malaria: plasmodium malariæ in the blood,* sequence of phenomena usually widely different, enlarged spleen, response to quinine, renal symptoms less prominent, jaundice later, facies of yellow fever absent, albuminuria delayed and hemorrhage rare. Only the irregular forms of malaria can by any possibility be confounded with yellow fever. *Acute yellow atrophy, Weil's disease* or *relapsing fever* lack the chief symptoms of yellow jack.

Seldom offers real difficulty.

MALARIA.—(Marsh miasm). **Definition.**—*An infectious disease*

caused by the hæmamœba or plasmodium malariæ and characterized in its common forms by periodic paroxysms, consisting of chill, fever, and sweating periods, and, by its response to quinine.

Historic Note.—One of the most ancient of diseases, malaria has been carefully studied in all ages, and when in 1880, a French army surgeon, Laveran, described its special organism, and Patrick Manson and Ross, a little later, demonstrated the role of the mosquito as intermediary host, it was found that little was needed to make all previously recorded and accepted observations fit the newer theory. Every one knew that the season of maximum prevalence of malaria was the spring, summer and early fall, that it prevailed most extensively in the tropics, that it tended to follow water courses and haunted the neighborhood of stagnant pools and fresh, brackish marshes, that salt marshes were free from it, and that sandy-bottomed *fishy* pools might abound without malaria. Its curious horizontal spread, the fact that dwellers in upper stories might escape it, the danger of night exposure, its rapid extension with the drift of prevailing winds, its disappearance under drainage and cultivation of land, all these things were thoroughly understood, even by the ancients, and each and every one corresponds to the life history of the mosquito. The carrier is the female anopheles, wide prevalence, a mosquito never absent from malarial districts, although anopheles may be present in non-malarial districts, the organism being absent. The world wide distribution of malaria makes it a disease of great importance, although generally speaking of low mortality.*

Mosquito intermediate host.

Conditions governing incidence.

Characteristics of the Mosquito.—There are probably over 500 varieties, the general appearance and character being too well known to require description. Like others of the order of Diptera, they lay eggs from which come the larvæ after two or three days of warm weather. These feed energetically on the water borne organic matter and are air breathers, a respiratory funnel being placed near the tail; hence they may be easily destroyed by the use of petroleum on the surface of their breeding places. The grown male insect is a vegetarian, the female seeks a mixed diet and sucks the blood, not alone of man, but of mammals generally, and, it is said, of birds, reptiles and even of fishes. The time required for full development is about 30 days in warm summer

Air breathing larvæ.

Easily destroyed.

* Fifteen thousand are said to die annually of malaria in Italy. It has been estimated that over 3,000,000 cases occur annually in Russia. Japan, India, China and the Philippines suffer greatly and the yellow races seem to have little or none of the immunity so evident in the Congo-black or pure negro, the Japanese, Chinese and East Indian being readily infected.

weather and the hatching process may be repeated several times during the season, one pair of insects producing millions of their kind.

The Special Characteristics of the Anopheles.—Length of palpi and proboscis about equal, the former four-jointed in the female and three-jointed in the male. The straight filamentous palpi are held parallel with the proboscis, the wings are often spotted, the abdomen is pilose with no scales, the legs are long, ending in dentate claws and the nucha has posterior scaly cornu.

Color in Relation to the Mosquito.—Nuttall has reported some interesting observations in regard to the color preferences of mosquitoes. He found that of the various colors ranging from white to black, the former was the least, the latter the most attractive. The observations are of distinct value in relation to the proper colors for clothing for dwellers in mosquito infested communities.

Dark colors attract.

Classification of the Organism.—As will be seen from the plates, the malarial organism is easily recognized and differentiated by blood examination and the expert observer can also closely predict the time of segmentation and recognize double or triple infection. The divisions correspond to the old clinical periods based upon the frequency and regularity of the seizures. *The tertian organism, requiring* 48 *hours for its development* brings about a paroxysm on each third day, the interval of apyrexia being one day. The *quartan, requiring* 72 *hours for its development, causes a paroxysm on each fourth day, with two days of apyrexia.* If two tertian organisms are developing, the paroxysm assumes a daily type; in the case of double quartan infection, attacks appear on two successive days leaving one day of apyrexia. If as rarely happens there be a triple quartan infection, the disease assumes the quotidian or daily type, as in the case of the double tertian.

Tertian

Quartan.

Double infections.

Estivo-autumnal Plasmodium.—This is irregular in its development and manifestations and is the variety which causes the severer, more chronic, malignant, or pernicious types of the disease. In the United States the tertian organism is exceedingly common; the quartan rare. The accompanying plates give a description of the various forms and show their progressive development at the expense of their erythrocyte host.

Quartan rare.

Examination of the Blood for Malarial Organisms.—Fresh blood on a warm stage gives the most satisfactory results in these examinations, but it is quite possible to detect them in the dried and fixed specimens, stained as in ordinary blood examinations. If the old triple stain be used, or the better stain of Wright, already described on page 380,

Fresh blood best.

DESCRIPTION OF PLATE III.

Quartan:—1, 2, 3, 4, 5 and 6, show the development from the hyaline form to the mature intracellular (6) and large extracellular (10) forms; 11 shows vacuolization of an extracellular form; 7, 8, 9, show segmentation stages; 12, the flagellate form.

NOTE:—In the tertian and æstivo-autumnal organisms the same phases are shown.

NOTE:—

A. Relative depth of color in the erythrocyte host, deepest in the quartan, lightest in the tertian.

B. Comparatively coarse, scant and dark granules of the quartan as compared with the tertian.

C. Tendency to shrinkage in the erythrocyte host of quartan vs. large pale host of tertian.

D. Peripheral arrangement of pigment in developmental stage of tertian and quartan followed by central grouping initiating segmentation.

E. Note greater regularity in quartan as compared with tertian forms.

F. Note relative number of segments.

G. Note greater density and clearer outline of quartan forms.

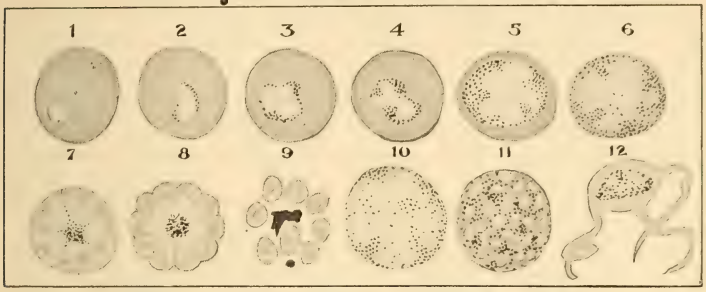
H. Star-like arrangement of pigment in early segmentation stage of quartan.

I. Relatively small flagellate forms of quartan and æstivo-autumnal.

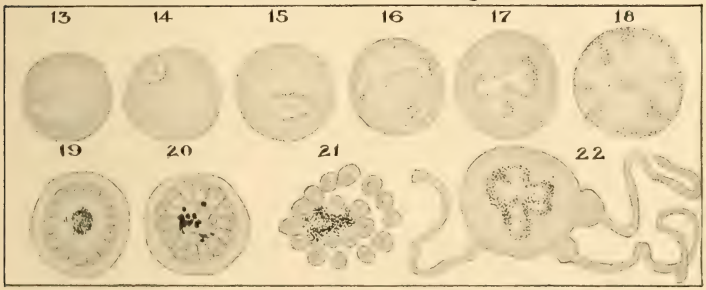
J. The peculiar ovals and crescents of the developed æstivo-autumnal form, its scantily pigmented spherical form (35) and the ring bodies of the early stage.

PLATE III.

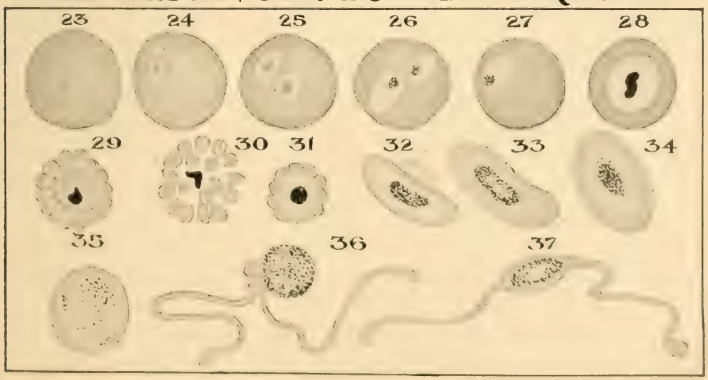
THE QUARTAN PARASITE.



THE TERTIAN PARASITE.



THE ÆSTIVO-AUTUMNAL PARASITE.



the organism will be found to have taken the blue. The common mistake born of inexperience lies in the failure to search *primarily* for *pigment*, and *in the readiness with which the unpigmented forms are confounded with artefacts*. In nearly all specimens taken at a proper time pigmented organisms may be found within the erythrocytes and pigment granules in the leucocytes. *In the tertian variety the pigmented forms appear from* 8–16 *hours after the preceding chill, and the large pigmented organism* is best obtained 8 hours before the paroxysm. It is useless to hunt for tertian or quartan forms if quinine has been taken for 24 hours or more.

Look for pigment.

Best time to search.

Symptoms.—The *tertian* and *quartan* forms are manifested by typical paroxysms whatever the frequency of their occurrence, and the order of events is as follows:—(a). *The cold stage:*—after a rigor lasting form ¼ to 1½ hours and occurring usually in the mid forenoon or early afternoon, the temperature, which rises with and during the chill, reaches a maximum (104–107 F.) and the cold stage is succeeded by (b) *the hot stage* which presents all the symptoms of sthenic fever which reaches its fastigium in an hour or two and declines to normal during the next 6–8–10 hours, its recession being marked by the coming of (c) *the sweating stage* during which all symptoms abate and the patient feels well. These attacks vary greatly in duration, those of the severe type lasting from 8–14 hours. The spleen enlarges if they are repeated on several days, and if long continued tends to become permanently hypertrophied (ague cake). In spite of the marked evidences of infection there is a normal leucocyte count or more frequently a leukopænia. Herpes labialis is a common manifestation, albuminuria may be present during the attacks or persist if they are frequently repeated, and the gastro-intestinal disturbances accompanying the fever may be profound. The *estivo-autumnal* form, due to the organism of that name, may at its onset be definitely periodic, usually like tertian but with longer paroxysms. Ordinarily, owing to the irregular development of the parasite crops, the fever tends to assume the remittent (bilious remittent) type and in such cases the temperature curve may be lower than in the fastigium of the regular form. Jaundice is common, gastro-intestinal symptoms and splenic enlargement marked. Under this head the most diverse types of malaria must be included, representing on the one hand the larval types, or dumb agues often manifesting themselves by persistent headache, neuralgia, general malaise, dyspepsia, anæmia, etc., and at the other pole the pernicious form of the disease encountered especially in the tropical and sub-

"Fever and ague."

Cold stage.

Hot stage.

Sweating stage.

"Ague cake."

Herpes and albuminuria.

Tertian.

"Dumb ague" sequelæ.

tropical countries. These assume various types, are often limited to a certain small area and are peculiarly fatal in the unacclimated.

Choleraic and comatose forms.

Classification of Pernicious Forms.—(a). *Choleraic.* Symptoms, low or slightly elevated temperature, vomiting, choleraic diarrhœa and stools, frequently a jaundice, cyanosis, feeble, rapid pulse, collapse and high mortality. (b). *Comatose form.* Usually preceded by a typical malarial paroxysm which is succeeded by stupor, hyperpyrexia, Cheyne-Stokes breathing and every symptom of collapse, stupor deepening into coma. (c). *The hæmaturic* or *hæmoglobinuric* form. This is characterized by the predominance of renal symptoms, associated at times with bleeding from the mucous membranes and includes undoubtedly the so-called "black-water fever." It is very fatal.

Black-water fever.

Usually estivo-autumnal.

Comment.—These pernicious forms are usually though not invariably associated with the estivo-autumnal organism; in the choleraic type masses of organisms may be found in thrombi of the intestinal vessels; in the cerebral or comatose form, in the vessels of the brain. The strong resemblance to yellow fever presented by these cases will at once be noted.

(For differential diagnosis see yellow fever.)

Chronic Malaria.—(Malarial cachexia). The chief characteristics of this form are the enlarged spleen, anæmia, sallow skin and persistent malaise. In many instances there is a tendency to chronic diarrhœa, dysentery or marked chronic gastro-intestinal disturbance of a vague and indeterminate type. In some cases there are accessions of fever, perhaps actual paroxysms occurring irregularly, and persistent and repeated examinations will usually reveal the estivo-autumnal organism. *Complications.* Persistent neuralgias, often regularly or irregularly periodic, persistent anæmia, gastro-intestinal difficulties and chronic nephritis are among the commoner complications and, more rarely, actual changes in the brain, spinal cord or peripheral nerves may occur, producing paralysis, ataxia or even symptoms of disseminated sclerosis.

Misleading symptoms.

Diagnosis.—The recognition of the regularly periodic form is not difficult but in general no accurate diagnosis can be made without an examination of the blood, the two elements of importance being the plasmodium and the leukopænia. Clinically dependence must be placed oftentimes upon the effect produced by quinine given in large doses and in solution. The differential diagnosis as regards typhoid and yellow fever has been discussed under those diseases.

Blood examination and therapeutic test.

RELAPSING FEVER.—Definition.—An acute contagious disease

DESCRIPTION OF PLATE IV.

Malarial Organism of types shown in plate but treated with Wright's stain.

QUARTAN PARASITE.

1. Non-pigmented form.

2, 3, 4. Young organisms showing chromatin bodies (red) and pigment granules.

5, 6. Full grown parasites, the former intracellular, the latter extracellular.

7, 8. Presegmentation forms.

9. Segmentation.

11. Normal blood cell.

12. Flagellate body.

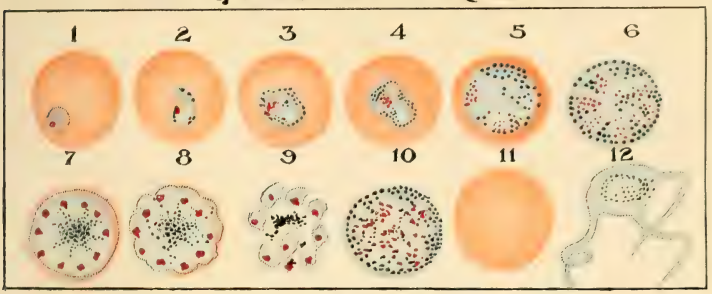
TERTIAN PARASITE. The forms are self explanatory in connection with the preceding description.

ÆSTIVO-AUTUMNAL FORM. Various ring forms are shown together with the characteristic crescents, Nos. 32, 33, 34.

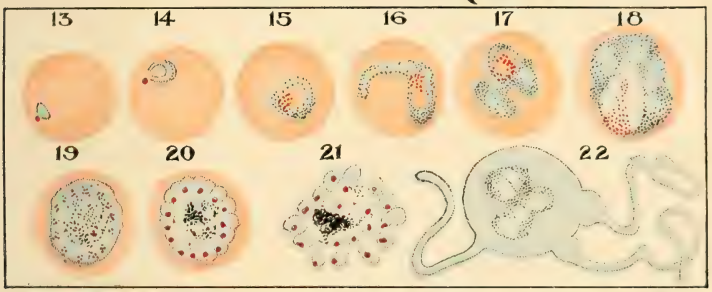
(In both this and the preceding plate the drawings are almost entirely based upon personal observation, though certain forms have been adapted from Thayer's valuable monograph.)

PLATE IV.

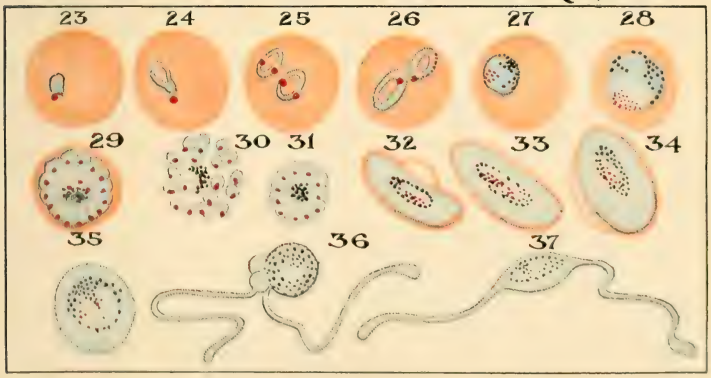
THE QUARTAN PARASITE



THE TERTIAN PARASITE



THE ÆSTIVO-AUTUMNAL PARASITE



due to the spirochæta Obermeieri, characterized by recurrent febrile paroxysms, each being of from 5–7 days, duration and associated with an acute onset and critical termination. **Etiology.**—The disease is one of filth and is highly contagious. The spirillum is unquestionably the cause of the disease but cannot be cultivated. **Morbid Anatomy.**—There are no special lesions, enlargement of the liver and spleen being common, together with catarrhal inflammation of the gastro-intestinal tract. **Symptoms.**—*The symptomatology is summed up in the definition.* It represents *repeated attacks of high fever of sudden onset terminating by crisis, alternating with afebrile periods of about the same duration.* The diagnosis is at once established by an examination of the blood which reveals the long spirilla which are in length about three times the breadth of the red cell. The usual complications of fever of this type may be present, i.e. pneumonia, nephritis, hyperpyrexia, jaundice, ophthalmia, and renal, gastric, or intestinal hemorrhage, yet in general the disease is uncomplicated and the prognosis favorable.

Fig. 167.—Spirillum of relapsing fever (spirochæta Obermeieri). Motile, flagellated, spirillum readily stained but not cultivated and Gram negative. The organism is usually longer than as shown in the illustration.

TYPHUS FEVER.—("Spotted fever," "ship fever"). **Definition.**—*An acute self limited infection characterized by sudden onset with high and continuous fever, terminating by crisis on or about the fourteenth day, and associated with a petechial eruption and a mottled skin.*

Historic Note.—This ancient disease was confounded with typhoid fever until the early part of the nineteenth century, and even the clear distinction made by Louis did not bring about its separation from typhoid until nearly half the century had passed. Once a common and fatal disease, it has now become one of the rarest and is limited to unsanitary, semi-civilized and densely populated countries or communities. Overcrowding, bad air and dirt have always been its chief promoters. It is highly infectious, but its micro-organism is unknown.

A banished scourge.

Morbid Anatomy.—There are no characteristic changes beyond those of an intense febrile infection.

Symptoms.—The symptoms characteristic of typhus fever are the onset of high temperature of a continuous character, preceded, or, at the outset accompanied, by chilling or actual chills with marked headache, backache, prostration and the rapid development of a typical "typhoid state," but the eruption is darker than in typhoid, and though

first appearing on the abdomen spreads over the whole body surface' and is accompanied by a curious mottling of the skin. The termination is usually but not invariably by a sharp crisis, relapse is rare and the complications are those attending any profound febrile infection with marked toxæmia.

Prognosis.—The mortality may reach 50% in old people, and varies from 10–20% in children and young adults.

MALTA FEVER.—(Mediterranean fever, undulant fever, "rock ever," Neapolitan fever, Gibraltar fever). **Definition.**—*An endemic*

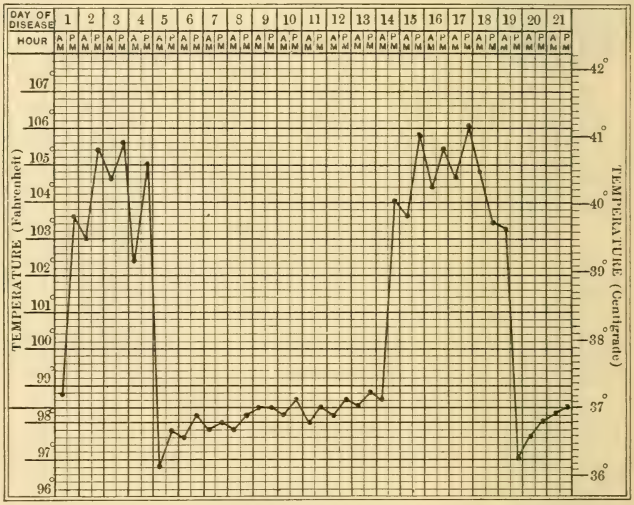


Fig. 168.—Clinical chart of relapsing fever showing the febrile movement upon the fourteenth day.—(From Wilcox's Fever Nursing.)

infectious fever due to the micrococcus melitensis (*Bruce*) and *characterized by long continued undulatory pyrexial periods and repeated relapses.*

Etiology.—The micrococcus melitensis, discovered in 1887, is a small oval or round coccus, staining readily though not by Gram's method, easily cultivated in bouillon, in agar-agar or gelatine, and showing an agglutination reaction similar to that of typhoid fever. By accidental inoculation, as well as by actual experiment, the specific action of the germ has been amply proven but the exact method of its dissemination is unknown. It is essentially a disease of summer, respects no age, is uninfluenced by sex and confers prolonged immunity.

Confers immunity.

Morbid Anatomy.—The pathological changes are not specific; there is general visceral congestion affecting the lungs, which in some cases show actual consolidation; the heart muscle shows degenerative changes, the liver and spleen are enlarged and in chronic cases show a fibroid change. There is moderate anæmia, and often a relative leucocytosis, the spleen contains the specific germ in quantities and shows an increase of lymphoid tissue.

History.—Unrecognized or at least undifferentiated, this disease has no doubt existed from time immemorial, along the shores of the Mediterranean and ancient writers are supposed to have referred to it under the head of malaria. Various observers have reported it in a

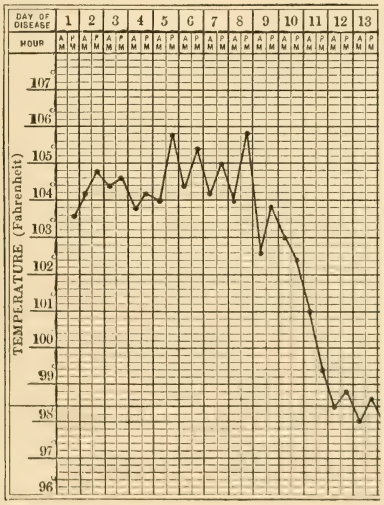


Fig. 169.—Clinical chart of typhus fever ending in recovery. —(From Wilcox's Fever Nursing.)

more recent period and it would seem to be widely distributed, cases having been reported from the East and West Indies, from Centarl America, and even in this country in isolated instances.

Period of Incubation.—The incubation period is from three days to three weeks, lasting on the average from ten to fourteen days.

Symptoms.—The onset closely resembles typhoid, the fever running in waves of three weeks' duration, repeated once, twice or many times with intervening periods of normal temperature. It may last for from

Resembles typhoid.

two or three weeks to two years, its average duration being about three months. As a rule, the first relapse is more severe than the original attack, being associated with chills, high intermittent fever and a diarrhœa which replaces an antecedent constipation.

Differential Diagnosis.—From *malaria* it is differentiated by the absence of the specific hæmamœba of that disease. From *typhoid* by the failure of the agglutination test of Widal. Aside from these procedures one must depend simply upon the differences in the general symptomatology, the temperature chart and the tendency to distinct relapses.

Prognosis.—Oddly enough, the disease furnishes but 2% mortality, the loss of time, the discomfort and interruption of affairs being the chief inflictions.

MILK FEVER.—(Milk sickness). This is a rare and frequently fatal disease limited to the southern and western part of the United States and is apparently derived from the ingestion of the uncooked flesh or milk of cattle suffering from "trembles."

Symptoms.—A short period of malaise is followed by vomiting and epigastric pain, fever, constipation and muscular tremors. The symptoms closely resemble ordinary ptomaine poisoning, and there may be active or typhoidal delirium, coma or convulsions.

MOUNTAIN FEVER.—There is no single disease to which this term may be properly applied. Most of the cases so denominated by the earlier writers were unquestionably unusual forms of typhoid fever.

MILIARY FEVER.—(Sweating sickness). In ancient times this disease was extremely prevalent and fatal throughout all Europe, but is now seen only in limited districts of Italy, France, Austria, Asia Minor, Germany and Switzerland. Following a short period of malaise the onset is marked by drenching sweats, persistent and recurrent, with from seven to ten days of high fever, and on the third day an eruption consisting of miliary red papules and tiny vesicles.

FOOT AND MOUTH DISEASE.—This is merely a *severe epidemic aphthous stomatitis* due to eating the flesh or milk of cattle affected with the disease of the same name. It is extremely rare, but furnishes a considerable mortality (10%).

FLOOD FEVER.—(Japanese river fever). This is a rare disease limited to a small area of the Island of Nippon, and afflicting only workers on the inundated land; amongst these it spares neither age nor sex. Its exact cause is unknown and it has no specific morbid anatomy.

Symptoms.—Ulcers in the axilla, groin or neck, associated with repeated chills, continued high temperature, moderate lymphatic involvement and profound toxæmia. A coarse papular eruption appears on the sixth day and disappears promptly after several days.

Prognosis.—The duration of the disease is from two or three weeks and the mortality varies greatly, running from 10–80% in different epidemics.

KALA AZAR.—(Black fever). This curiously obscure and deadly disease has for some years prevailed in a limited district of Assam, having commenced its onward march at Rungpore in 1887. It extends along lines of communication at the slow rate of 14 miles a year, its visitation lasting for five years and the natives believe that one year is required to render safe any house in which it has appeared. It spares no age and neither sex, and what is more peculiar attacks the acclimated quite as readily and violently as the stranger. Its mortality is frightful, being put at 96% by Manson, and its victims may linger on for a year or more.

Etiology.—The causative factor is unknown. It has been regarded as malaria, uncinariasis, modified Mediterranean fever and trypanosomiasis.*

Symptoms.—The high fever, often preceded by vomiting or chill may assume either a remittent or intermittent type, lasting one, two, four or six weeks; then follows an apyrexial period, then an exacerbation, and so on for months, when the fever becomes persistent though remittent and profuse sweats characterize the remissions. Emaciation is extreme, liver and spleen enlarge early, profound anæmia supervenes, and a dirty gray pallor and brittle dry or falling hair add to the striking clinical picture.

A ghastly clinical picture.

Termination.—The patient dies ordinarily from an intercurrent pneumonia, dysentery or exhaustion.

MEASLES.—(Morbilli). **Definition**.—An acute highly contagious febrile exanthem of childhood of unknown causation, conferring immunity, and characterized by marked catarrhal symptoms of the respiratory tract and a peculiar eruption.

Etiology.—Children under 12 years of age are remarkably sus-

*The disease in many respects lends itself to each theory, though the fact that agglutination of the micrococcus melitensis has been shown by Bentley's work would seem to strongly support the Malta fever theory. The observations by Rogers made during the last year are interesting but not conclusive.

ceptible,* and the winter and spring are its chief seasons. *Contagiousness. Practically every exposure in children under ten results in an attack* and as it spreads by proximity, contact and fomites, and unfortunately in both the early and later stages (diminishing only with established convalescence), few children escape infection and its virulence increases with the age of the person attacked. The mortality is terrific amongst the poorer tenement classes but in private practice it should not exceed 5% amongst well nourished children.†

Few escape contagion.

Morbid Anatomy.—Merely that of the complications and catarrhal inflammation of the respiratory tract. Incubation one to two weeks, usually eight to twelve days.

Symptoms.—The presence of a *marked coryza, injected conjunctivæ, photophobia and indications of slight fever may be the only signs noted at the onset.* Chill or decided chilliness is less common.

Fever.—The fever rises steadily, reaching its maximum (103–105° F.) with the *appearance of the exanthem* (4th day). Its subsidence begins as the rash commences to fade, and the normal is attained, either by crisis or a lysis occupying from two to four days.

Important fever curve.

The Rash.—*White tipped, red spots* (Koplik's spots) on a congested buccal or labial mucous membrane appear early in 90% and the mucous membrane may show the true eruption before the skin is affected.‡ *At the end of the third or during the fourth day, and at the height of the fever, the general rash becomes manifest as a dusky mottling*, becoming more distinctly red though still dusky, *and at the end of 24 hours* (fifth day usually) *palpably papular* and "*shotty.*" It appears first at the *margins of the hair and in the region of the ears* and the forehead and gradually spreads over the forehead, neck, face, chest, trunk and lower extremities, often assuming a crescentic arrangement and reaching its acme and fading in its first location before reaching its maximum in the later ones, so that *cases are likely to present rashes of varying stages of development in the different portions of the body.* Desquamation, fine and branny follows and lasts for about one week.

Character and distribution.

Desquamation.

Duration of Stages.—*Period of incubation*, 1–2 *weeks. Period of*

* Continued prevalence seems to confer a partial immunity for the disease is terribly fatal when introduced into countries previously exempt.

† In favorable climates much less.

‡ Koplik's spots are rose red with a punctate bluish white centre tending to spread and multiply as the disease progresses and giving place to general redness and a multitude of spots at the time that the general body eruption is developed.

invasion, 3–4 days. Period of developed rash, 1 week. Period of desquamation, 1 week. Important data.

Complications.—Broncho-pneumonia is the most common and serious one, causing death in 30–40% of such cases in the public services, and even in private practice furnishing a heavy mortality. The disregard of this disease manifested by the laity is deadly in its effect, as children are exposed to pneumonia and other complications from sheer ignorance of its high mortality.* Broncho-pneumonia is an especially deadly complication in sucklings. Severe conjunctivitis and keratitis occur especially, often in those poorly nourished and badly cared for, and the same may be said of severe gastro-intestinal disturbance, stomatitis or even gangrenous processes. The eyes may remain sensitive for a long period. Pertussis is frequently associated with measles, tuberculosis is a not infrequent sequel, and the milder degrees of otitis media and affections of the nasal structures are not infrequent. *The Urine.* Transient albuminuria is not uncommon, and Ehrlich's diazo-reaction is invariably present. *Blood:*—Leukopænia or a normal count is the rule.

An underrated ailment.

Sucklings.

The eyes.

Diazo.

Diagnosis.—*The combination of a febrile period of three full days associated with marked coryza, Koplik's spots* and the *peculiar eruption* serve to *distinguish the disease from drug and serum rashes and rubella.* The temporary measle-like rash of the preliminary stage of smallpox must always be borne in mind. Great variations in the disease exist; it may be trivial or virulent, febrile or afebrile, with a very slight or absent exanthem. The eruption may be hemorrhagic (black measles) and cases may, from the start, show predominance of nervous or pulmonary symptoms and the lungs should be examined carefully, at every visit. A case of *persistent measle-like rash* may prove a syphilis.

Look out for smallpox.

Syphilis.

MUMPS.—(Parotitis, epidemic parotitis). **Definition.**—*An acute epidemic, infectious, immunity conferring, highly contagious but usually trivial, disease of childhood of unknown causation characterized by inflammation of the parotid gland and mild systemic symptoms.*

Highly contagious.

Etiology.—Children under four and adults are but slightly susceptible. The disease is transmissible in any stage, by contact, contiguity or fomites.

Incubation Period.—48 hours to three weeks. *Duration.* Active symptoms about one week, gradual subsidence.

*The public services of the great cities furnish shocking examples, and the author remembers a little girl who was found dying of broncho-pneumonia *facing the bright sunlight* with two eyes whose corneas had melted away like mist. "She had but a touch of measles," said the mother.

Morbid Anatomy.—An acute parenchymatous inflammation of the parotid gland.

Symptoms.—*Pain and swelling*, usually bilateral, but often single, *in the parotid region. Fever* usually mild (102° F.) and of short duration. *Dry mouth, intolerance of acids.*

Adults chiefly.

Complications.—In the comparatively rare cases in adults unilateral or rarely bilateral orchitis is a frequent complication (20–30%), though rare in children. Suppuration of the gland by infection through Stenon's duct is a rare development. In the female, ovarian congestion or inflammation or painful swelling of the breasts may, rarely, be found.

Ovaritis. Orchitis. Mastitis.

SCARLET FEVER.—(Scarlatina.) **Definition**.—*An acute infectious and highly contagious fever of children of unknown causation, conferring immunity and characterized by a sudden onset, the prompt appearance (second day) of a punctate bright scarlet exanthem, terminating in desquamation and showing a marked tendency to inflammatory involvement of the kidney, middle ear, throat and cervical glands.*

Etiology.—The disease is conveyed chiefly by *direct contact and fomites*, less often by proximity, the secretions being, according to late investigations, more potent than the desquamated scales. In hospitals severe and complicated cases should be kept away from the mild and uncomplicated. It attacks chiefly children under 16 years, but adults enjoy only a relative immunity.

Hospital cases.

Prevalence.—It prevails throughout the year, but chiefly in the autumn and early winter months and covers nearly the whole globe.

Mortality.—The general mortality varies from 10–15%, and in children under 6 years of age, it may reach 25 or 30%. As in other diseases of this group the mortality varies greatly in private as compared with public service practice, and in weaklings as compared with the sturdy. A damp cold climate seems to increase the severity and frequency of complication, and hence the mortality.

Age, physique and climate.

Symptoms.—With or without antecedent malaise there is (a) an abrupt onset, usually marked by *vomiting* with or without nausea, and commonly with *sore throat*, indeed the latter symptom may be dominant. (b). *Fever*, 100–105° F. at onset, *diminishing by lysis with the complete development of the rash* and reaching normal in a week or ten days, unless complications occur. (c). *The rash. This appears in from 12 to 24 hours, first on the chest*, and spreads with great rapidity. *It is really punctate*, the color being deepest about the hair follicle, *but it appears diffuse and is commonly described as a lobster-like redness* which spreads over the whole body in a few hours. As in every other

Abrupt onset.

High initial fever.

Early rash.

eruptive fever, *it varies greatly in intensity*, in some cases escaping superficial observation. It should be remembered that *it is most intense about the flexor aspect of the joints, the groin and inner aspect of the thighs, and on the anterior surface of the upper chest.* In marked cases it is unmistakable and *the fact that about the upper lip it is often almost absent gives a characteristic facies.* It fades in from three or four to seven or eight days and a *remarkable desquamation* follows, quite unlike that of measles. *The skin peels and may come away in large sheets.* Exfoliation may be complete in a few days, or last for three or four weeks, and the scales are infectious, though probably less so than has been believed. The order of desquamation is usually, chest and neck, 4th–7th day; hands 12th–14th; feet 14th–21st. (d). *Rapid pulse.* Excessive rapidity may be present *before the appearance of the rash* and persist throughout the active stage. (e). *Strawberry tongue*, light, white or grayish furring punctuated by red and prominent papillæ is a somewhat prominent feature.

Points of election.

Peculiar facies.

Lamellar peeling.

Rapid pulse.

Strawberry tongue.

Complications.—*The danger in scarlet fever lies in its complications* and of these *nephritis* is most to be dreaded. This when present takes the form of an *acute nephritis*, developing usually at the end of a week. Mere febrile albuminuria is common and sharp congestion may occur and promptly subside. *A hard pulse should direct attention to the kidneys, the urine should be measured and a specimen examined at least once in 24 hours*,* and the patient should be carefully guarded from exposure to cold and wet *for some time after* the disappearance of all active symptoms of scarlatina.

Nephritis.

Safeguards.

Suppurative otitis media is another dangerous complication, resulting oftentimes in mastoid abscess, septic meningitis, general sepsis and permanent deafness. The throat should be kept as clean by sprays and gargles as the circumstances permit, and the ears should be watched carefully, especially in infants. *Swelling of the cervical glands* is common and may result in extensive infiltration and suppuration.

A cause of deafness and meningitis.

Pharyngitis and Tonsillitis.—These may be extreme and a true or pseudo-diphtheritic membrane may be present. The occasional coincidence of scarlatina and diphtheria is not to be forgotten. *Endocarditis, pericarditis* and *myocarditis* frequently result and the heart should be carefully auscultated at each visit. *Parotitis, septic arthritis, broncho-pneumonia, lobar pneumonia, pleuritis* and *empyema* are rare complications but *active delirium* and even *convulsions* may be present.

Diphtheria.

Other complications.

*The early administration of urotropin, and the free use of Vichy and cream should diminish the frequency of this dreaded complication.

Diagnosis.—*Sudden onset, vomiting, fever, sore throat, a typica rash and a rapid pulse are unmistakable,* but many cases arise which tax the resources of the cleverest clinician. *Doubtful rashes may be made plain by the hot pack or hot bath. Excessively rapid pulse* is important in connection with other suggestive symptoms. The *hard pulse suggests nephritis.* A *secondary rise in temperature always suggests a complication.* Cases occur with no discoverable rash and no fever, and these may transmit the disease in its typical form to other children.* The diazo-reaction is present.

Doubtful rashes.

Hard pulse.

Spurious Rashes.—Unfortunately roseolas are common and often due to trivial causes. In differentiation one must bear in mind that the scarlet fever rash appears first on the chest, is seldom unassociated with sore throat, rapid pulse, enlarged lymphatics and decided fever and desquamation, following the order given in a preceding paragraph. Rubella should never be mistaken for it as the eruption appears first on the face and is macular. *Indigestion.* Shell fish and strawberry rashes lack the other essential features. *Drug rashes* may be traced to their cause and lack cardinal signs. *Septic rashes* are known by their association with other signs of sepsis, onset, etc. *Syphilis.* Scarlatina-like rash is rare in syphilis, and the other concurrent signs make the diagnosis. *Febrile Roseola.* Simple (non-epidemic). Roseola exactly simulates mild scarlatina, but its benign character and the history of repeated attacks may serve to distinguish it. All suspicious cases should be isolated, and no chances taken.

Location and associated symptoms.

Febrile roseola.

RÖTHELN.—Definition.—*A trivial acute contagious exanthematous disease of unknown causation conferring immunity and chiefly affecting children.*

Symptoms.—After an incubation period of from 7–21 days the disease begins with slight malaise, trifling fever and on the second or even first day the rash appears *on the face,* spreading rapidly over the body, the old areas fading as it extends. Either the measles or the scarlatina rash may be closely simulated but the disease protects from neither of these ailments and the trivial symptoms readily distinguish it. Neither catarrhal symptoms nor sore throat are marked but swelling of the post-cervical glands is common. There is practically no mortality or serious complication and the diazo-reaction is usually absent.

Trivial symptoms, evanescent rash.

DIPHTHERIA.—(Cynauche maligne, putrid sore throat, suffocative angina, membranous croup).

*Any man is negligent who fails to satisfy himself each day that the urine, heart, throat and ears are unaffected.

Definition.—*An acute highly infectious, contagious and profoundly toxic disease, due to the Klebs-Læffler bacillus, characterized by the development of a nasal, pharyngeal or laryngeal false membrane and especially affecting young children.*

Dissemination and Distribution.—The disease is well nigh universal and is readily disseminated by contact, contiguity (cough dissemination), clothing, drinking utensils, pencils, school books, and intermediaries such as parents, nurses, physicians and certain persons who undoubtedly carry the germ but are themselves unaffected.* Such may carry the germ for months or years. So also food stuffs, pet cats and dogs, room or street dust may convey it.

All channels available.

Infectious individuals themselves well.

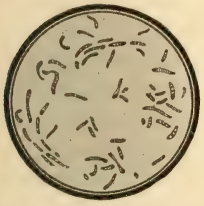


Fig. 170.—Diphtheria bacillus (bacillus diphtheriæ) of Lœffler. Pleomorphic, non-motile, non-sporogenous, non-flagellate, non-liquefying, non-chromogenic, aërobic, readily cultivated and stained by all methods. F. F. Wesbrook has described (a) a virulent type with clubbed extremities and polar granules, (b) granular type, (c) barred type, (d) solid type.

Morbid Anatomy.—Aside from the false membrane itself the interest centers about the heart, nervous system, and kidneys which are the chief sufferers from the diphtheria toxin. The most important conditions are myocarditis (the most frequent complication and cause of sudden death), laryngeal stenosis and peripheral neuritis. Anterior poliomyelitis or even hemorrhages into the spinal cord are common and the cranial nerves are quite frequently affected. Congestion of the kidneys is almost invariable and acute nephritis not uncommon. The spleen is hyperæmic, the lungs not infrequently congested or the seat of broncho-pneumonia.

Organs chiefly affected.

Symptoms.—*No disease is more variable in its symptoms and the modern physician has learned to expect diphtheria without marked fever or malaise and even in the absence of marked throat symptoms or the presence of an apparently frank and relatively innocent tonsillitis, while on the other hand what appears to be a true membrane upon the tonsils and pharynx may prove non-diphtheritic.* Ordinarily there is *chilliness* and *general malaise*, the *fever rising to* 102–103° during the first day, and there is *nearly always sore throat*, and *what is far more important as an early sign* and in dubious or larval cases, *enlargement of the glands at the angle of the jaw*. If the child has been sick for some days its appearance and evident weakness may be out of proportion to the tem-

Diphtheria without membrane and *vice versa*.

A valuable sign.

*In a case recently observed the symptoms were practically nil though culture was positive and yet a sister contracted by contiguity a virulent diphtheria.

perature and local signs, yet here again exceptions occur and extensive membrane and marked glandular involvement may co-exist with slight systemic disturbance. *The pulse is usually rapid* and not infrequently irregular and irritable from the onset but its more significant variations appear as a rule later in the disease when it may be *very rapid, very slow, or show decided arrhythmia.* It is moreover *weak and compressible* in any established case *unless there be some decided renal involvement. The appearance of the throat is variable and it may be that of a simple follicular tonsillitis, or more often shows a definite patch or extensive membrane spreading to the faucial* pillars and perhaps to the *uvula, naso-pharynx* or *even the larynx. It is gray or drab* (mouse color), *dirty and usually shaggy and if removed leaves a raw bleeding surface* which it quickly re-covers. The spread of the membrane is sometimes astonishingly rapid and even on the physician's first visit it may have reached an extreme development. The nervous symptoms of the early stage may be nil or consist of restlessness or delirium. In the later stages a state of *profound nervous depression* may be evident with or without low delirium, stupor or terminal coma. Attention should be paid to the *nose* and *larynx* no less than to the throat in every case suggesting diphtheria, as *the membrane may be limited to these regions, and the utmost care and caution should be exercised in allowing the child to assume the sitting posture* and *getting it out of bed during convalescence.* So also *the urine should be frequently examined and the heart auscultated and percussed at each visit.* The wise physician will use antitoxin promptly in dubious cases without waiting for a laboratory report, though these are now furnished with remarkable promptness. Nasal diphtheria may at times only be detected by a rhinoscopic examination, laryngeal cases by the laryngoscope, but the evidence of related stenosis in either case, together with the symptoms of profound exhaustion which almost invariably accompany them will point the way In the former an irritating nasal discharge is common. *Membranous croup has properly been shelved* by modern methods of diagnosis *and replaced by laryngeal diphtheria* which in 99% of such cases is the proper descriptive term. The symptoms of *dyspnœa* and *suffocation from laryngeal membrane* do not need description but no more terrible picture is seen in medicine and it of course demands *immediate* recourse to intubation, and that failing, to tracheotomy. *Both the nasal and laryngeal forms yield a higher mortality,* in the latter not only because of the obstruction, but more because of the higher degree of toxæmia associated with these two varieties.

Heart symptoms.

The throat.

The membrane.

Nervous system.

Nose and larynx.

Antitoxin.

Stenotic symptoms.

"Membranous croup."

Complications.—*Myocarditis* is the most common one and accounts for many cases of sudden death. In controllable children efficiently treated with large doses of antitoxin it should occur less frequently than it now does. The uncontrollable ones practically *commit suicide. Endocarditis* is not uncommon and *in any event a weak, rapid, excessively slow, irritable, or irregular pulse demands the utmost care and caution not only during the attack but long after it.*

Sudden deaths.

Suicides.

Complicating Paralyses.—These may be extreme and severe, or localized and relatively slight. They most commonly affect the palate, occur during convalescence and *almost invariably recover completely.* The complication may be expected in between 10 and 15% of all cases even though treated with antitoxin. *Diaphragmatic paralysis* is a rare event but may cause death.

All recover.

Profound hemorrhages are unusual and nephritis may, but seldom does, prove persistent. *Anæmia* should receive attention as soon as the membrane has disappeared and not be postponed until late in convalescence.

Differential Diagnosis.—The symptoms described enable any one to make a reasonably sure diagnosis in most cases but this is *always to be controlled by cultural methods. The most important points are the membrane, its distribution, its common tendency to spread rapidly and involve adjoining surfaces, the decided evidence of toxæmia, glandular swelling and, nasal or laryngeal stenosis in certain cases.* In taking a culture due regard must be had for the situation of the membrane and an attempt made to reach the larynx or nasal passage with the swab if the pharynx is free from exudate. *Follicular Tonsillitis.* The onset is more severe than is usual in diphtheria, the exudate is likely to be purely follicular (spots) and to show less tendency to rapid spread, is lighter, clearer and less adherent, does not as a rule extensively involve surrounding structures and the glandular involvement is less marked. *If operations have been recently performed the wounded surface may be covered with a membrane closely resembling that of diphtheria and lead to error.* **Vincent's Angina.**—To Prof. Vincent we owe our knowledge of an angina which, ordinarily assuming the form of an ulcerative or ordinary tonsillitis or pharyngitis, may closely simulate diphtheria. Two organisms are described, one a fusiform bacillus, the other a spirillum.

Culture, the only means.

Follicular tonsillitis.

Wound membranes.

Prognosis.—There are differences in epidemics and the surroundings, previous health and resisting power of the individual child cut a considerable figure, *but practically, the whole outcome depends upon the promptness with which antitoxin is administered and the adequacy of*

the dose. It is impossible to get accurate statistics but we know that without antitoxin the mortality reaches from 50–60%, while with antitoxin in private practice where cases are promptly seen and children well nourished it may not exceed or reach 5%.

SMALLPOX.—(Variola). (A.S. *pock*, bag or pocket).

Definition.—*An acute infectious and highly contagious disease conferring immunity, and characterized by a definite mode of invasion and a peculiar eruption.*

Smallpox vs. Great pox.

Historic Note.—Smallpox was so called originally in contradistinction to great pox (syphilis.) It has existed for thousands of years in Eastern countries, was introduced into Europe in the eighth century, and by the tenth had become so general throughout Europe that prior to the day of Jenner, few persons failed to show on the face its characteristic pits.* It was first brought to the American continent by the Spaniards in the sixteenth century, and to Massachusetts in 1633. The aborigines and negroes seem to be especially susceptible and the disease flourishes in its most virulent form on virgin soil. Once so general and fatal it has in our day become a comparatively rare disease with a low mortality. For this we have to thank that great and courageous physician, Edward Jenner, who, being convinced of the identity of cowpox and smallpox, in 1796 vaccinated, and six weeks later inoculated with smallpox virus, a boy of eight, reporting the success of his experiment in June, 1798. To appreciate his courage one has only to read the journals of that period in which he was most heartily denounced and abused. Prior to this time, Lady Wortley Montague had become convinced of the value and efficacy of inoculation as practised in the far East and succeeded in her efforts to introduce this procedure into England.† Unfortunately, the extreme care characteristic of Eastern nations in choosing a proper age, season and state of health was not observed by their English imitators, with the result that the good accomplished was over-balanced by the extreme mortality and disease incidence engendered. The Asiatic races had for centuries successfully practised inoculation upon a most rational basis, nevertheless the disease is still a scourge in India, where effective vaccination is rendered extremely difficult by the attitude of the people, who believe smallpox to be a form of purification sent by the gods and who object to the use of cows for the production of vaccine.

Formerly inevitable.

A medical hero.

Inoculation.

* An old writer has said "from smallpox and love few escape."

† Coincidently Cotton Mather and Dr. Boylston braved adverse public opinion and mob violence in Boston in the same cause.

Etiology.—The cause of smallpox has not been established. In 1892, Guarnieri described a parasite (cytoryctes variolæ) and Councilman and Calkins have attempted to show the stages in the development of the organism but its specific nature remains unproven. Organism unproven.

Age and Sex.—No age and neither sex is exempt, and rarely, intrauterine disease may exist if the mother be affected.

Morbid Anatomy.—The appearance of the patient who has died of smallpox is sufficiently indicated by the symptomology. The lesions of the viscera are essentially those of profound toxæmia and high fever.

Contagiousness.—*The infectious element exists in the secretions, excretions, pustules and probably in exhalations from the lungs* and the skin conveys it through the medium of the dried scales which are readily converted into dust. Though contagious at all stages, the frequent exposures without infection indicate that its early periods are comparatively inocuous. The contagium is difficult to destroy and remains active for long periods, hence the radical measures employed in the disinfection of rooms in infected houses. The question of conduction through the air is still *sub judice* but has much to support it. It may be conveyed not only by the clothing, but by room dust, by rats, mice, flies or vermin, *and the patient ceases to be an infective agent only when the last bit of desquamating skin has been removed. The severity of the disease bears no relation to that of the case from which it is derived.* Remarkably transmissible. Early stages slight. Fomites and carriers.

Individual Susceptibility.—This varies greatly, some being immune, even though unvaccinated and those sufficiently vaccinated being either wholly immune, or subject only to mild attacks.* Natural and acquired immunity.

Varieties of Smallpox.—There are six distinct forms varying greatly in severity, namely, *varioloid, discrete, confluent, hemorrhagic, malignant and verrucose.* Six forms.

Period of Incubation.—From seven to fifteen days.

Characteristic Symptoms.—(a). *Sudden onset,* often with chills, or in children, convulsions. (b). *Headache,* intense and frontal; this corresponds to the congestion of meninges found at autopsy. (c). *Severe backache* (congestion of the membranes of the spinal cord). (d). *Vomiting.* (e). *Severe pain in the limbs.* (f). *High initial temperature* Tearing backache.

*Since the Spanish-American war, a mild form of smallpox has prevailed in this country and spread widely, especially in country districts, because of its close resemblance to chickenpox, and further because the prolonged immunity resulted in careless disregard of the necessity for vaccination. According to some observers the light form is gradually becoming more virulent.

Peculiar fever.

(103–105 F.) *followed by a marked recession and a high secondary rise associated with suppuration of the vesicles.* (See chart, p. 57.)

Note.—*The pains are more severe than in any other definitely eruptive disease* and in the presence of an epidemic these symptoms must be given great weight though they are in no sense indicative of the severity of a given case.

Eruption.—Rarely the disease occurs without any true eruption or a tardy one, and in from 10–20% of the cases misleading initial rashes occur during the first two days. These assume one of six forms:—

Important *initial* rashes.

(1). Erythematous. (2). Urticarial (very rare). (3). Morbilliform. (4). Scarlatiniform (3 and 4 are extremely misleading). (5). Purpuric. (6). Astacoid (lobster-like). It is said that this sixth variety indicates a fatal issue. These eruptions may be mixed, are distinctly localized and limited to (a) the inner *surface of the thighs*, (b) *the lateral thoracic areas*, (c) *the lower abdomen*, (d) *the axilla. They persist until the appearance of the true rash.*

Localization.

The Typical Eruption.—On the *third* day *of the disease bright red macules* appear, readily blanched by pressure, these rapidly becoming hard elevated "shot like" papules. *At the end of 24 hours* these show *vesicles* and the *vesicular stage reaches its full development* usually by *the third or fourth day of the eruption.* The vesicles rarely exceed one-fourth of an inch in diameter, contain a milky fluid and are surrounded by distinct though narrow areolæ. *By the fifth or sixth* day depression of the apices of the vesicles is noticeable. This *umbilication is usually general* by the end of the first week of the disease (seventh or eighth day). *Almost coincidentally the vesicles become distinctly purulent (pustulation being general from the eighth to the tenth day and associated with a secondary temperature rise to 102–104+°F.). The whole process is completed usually by the end of the twelfth day.* By the *fourteenth desquamation should commence and be completed at the end of three or four weeks.*

Macules, papules, and vesicles (3d–4th day).

Umbilication (5th–6th day of rash).

Pustulation 6th–12th day of rash.)

Secondary fever.

Desquamation.

Parts Affected.—*The eruption chooses by preference the portions exposed to light, chafing, irritation and injury, and upon them reaches its highest development.* It usually *first appears on the face and near the border of the hair*, yet the *mucuous membrane may be still earlier involved*, the rash appearing on the buccal mucous membrane, tongue, soft palate, pharynx and even upon the stomach and rectum in the virulent cases and *in none do the mucous membranes escape.* The wrists are also affected early, and like syphilis but unlike varicella *it involves the palms of the hands*, a point of considerable importance in

First on mucous membranes; next, the face.

Palms affected.

differential diagnosis. The involvement of the larynx may cause distressing cough and even fatal edema.

Confluent Smallpox.—In this form all symptoms are intensified and in the stage of pustulation the patient presents a frightful appearance and a foul and *characteristic odor*. The face is a swollen and unrecognizable mass of fetid sores, though on the body and legs the eruption often remains discrete. Odor.

Hemorrhagic Form.—(Purpura variolosa), (black smallpox), (variola hemorrhagica). As indicated by the name, the eruption is distinctly hemorrhagic in type, the patient presenting a frightful appearance. In some cases hemorrhage occurs from any or all mucous membranes and *it has proven fatal in from three to four days after the onset of hemorrhagic symptoms* though in others of milder type the hemorrhages are limited to the vesicles and pustules. Severe vs. Mild.

Malignant Smallpox.—This is characterized by *low fever, scant or absent eruption, profound asthenia and a fatal issue* with or without hemorrhage, death occurring between the third and seventh days.

Variola Verrucosa.—This varies from the other forms only in leaving a warty growth after desiccation and hardly deserves separate classification.

Varioloid.—*This is an attenuated smallpox*, modified by vaccination, natural immunity, or a previous attack. It differs from true variola in the following particulars:—(a). *Short duration.* (b). *Irregular or incomplete eruption which runs a rapid course.* (c). *The absence of any marked secondary temperature rise.* (d). *The early completion of desquamation.* The *initial symptoms* are usually *but not necessarily mild* and the eruption may or may not be limited to the face and hands. Mild symptoms.

Complications.—These are easily inferred from the symptoms and pathology of the disease, being chiefly connected with the suppuration and profound toxæmia. It suffices to name them:—Edema glottidis, necrosis of cartilages, broncho-pneumonia (sometimes initial), lobar pneumonia (rare), pleurisy (not uncommon), pseudo angina, diarrhœa (especially in children). albuminuria (true nephritis rare), orchitis, ovaritis, parotitis, abortion, fatal delirium, myelitis, neuritis, pyæmic abscess, general tuberculosis, local gangrene, arthritis (suppurative or not), bone necrosis, otitis, iritis, keratitis, purulent conjunctivitis, phlebitis, etc.

Prognosis.—Absolute figures are out of the question. Assuming a severe epidemic, from 25 to 50% of the unvaccinated will die; amongst In unvaccinated, great mortality.

the vaccinated (varioloid) the mortality will vary according to the efficacy of the primary vaccination and its proper repetition (see vaccination), and with the age and physical condition of the person attacked. The *mortality in all unvaccinated cases is exceptionally great in infants and young children. Efficiently vaccinated persons are practically immune and of those attacked the mortality should not exceed from five-tenths of one per cent. to one per cent.* Of the hemorrhagic and malignant cases, practically *all die within the first week, and the same is true of unvaccinated children under one year of age. Dissipation and pre-existing disease* increase the virulence of the primary attack and its mortality, and *the more profuse or confluent the facial eruption, the greater is the death rate.* Pitting is unusual nowadays in the ordinary forms.

In vaccinated, negligible.

Disease type, age and physique.

Pitting.

Diagnosis.—*In the presence of an epidemic, cases presenting severe pain in the head, back and limbs, and vomiting, must at once be under suspicion.* Indeed lacking an epidemic they are still important and the greatest care should be observed when such symptoms are associated with the suggestive and *peculiarly located initial rashes. The typical eruption is unmistakable and the course of the fever most significant,* viz.:—appearing on the third day, being primarily high and dropping to normal, or in severe cases, to 100° F. or 100.5° F., *only to reappear with pustulation.* For differential diagnosis see chickenpox and syphilis.

VACCINATION.—The origin of vaccination with cowpox virus as the supplanter of inoculation from smallpox pustules is, as before stated, directly traceable to the keen observation and rare courage of Dr. Edward Jenner, who first proved its efficacy on May 14th, 1796. As a result of his brilliant work millions of lives have been saved, and smallpox has lost its terrors in every intelligent and civilized country. Few remain who deny the efficacy of vaccination, but they are of the type who would insist that the sun moves around a stationary earth. Wherever vaccination is required and *enforced* by law, smallpox is a negligible factor.

A boon.

Antivaccinationists.

Extent and Duration of the Protective Influence.—It should be clearly understood that to be effective and efficient vaccination must be repeated at certain intervals. *First performed in infancy, it should be repeated at the age of puberty, and in the presence of any especial liability to exposure.* The experience of every physician shows *almost absolute immunity* to be the result of intelligent and repeated vaccination, however direct the exposure. Furthermore, *the disease if contracted by*

Revaccination necessary.

Immunity results.

a vaccinated person is almost invariably the mildest possible (varioloid).* The false idea that one vaccination in infancy or childhood is absolutely or approximately protective throughout life is altogether too widespread amongst the laity and much of the prejudice against vaccination, aside from that depending upon pure ignorance, obstinacy, or more or less ingenious misrepresentation, is the result of the vaccination of the earlier days when the virus was usually taken from the vaccinated human being, and the operation performed with scant regard to the danger of septic infection. The antivaccinationists still talk of the *transmission of syphilis* as if an innocent heifer could acquire and transmit a disease of this peculiar nature. *It should be remembered that even though a person has been actually exposed to smallpox, vaccination will ordinarily either confer complete immunity, or greatly modify the disease,* it being understood that the time elapsing between exposure and development shall have been sufficient to allow of some action on the part of the vaccine. As between cases *of even doubtful vaccination* and the unvaccinated, the ratio of mortality is as one to four.

The "conscientious objector."

The maligned heifer.

Vaccination *after* exposure.

Preparation of Vaccine.—The material for vaccination is now invariably obtained from the heifer, and the so-called humanized lymph should not be used, except in emergency. Various reliable firms that prepare this substance maintain farms and conduct their inoculations and the recovery of the virus under the most perfect sanitary conditions. The source of the lymph is the vesicle produced by the inoculation of the udder of the cow. This is allowed to dry on sterilized ivory points or quills, or is treated with glycerine, and collected in sterilized glass tubes, so contrived as to permit their use without danger of contamination.† *Method.* The region of the deltoid insertion of the arm or the outer surface of the thigh or calf should be thoroughly cleansed with alcohol (cologne, bay rum) and washed off with boiled water, then lightly but rapidly scratched by a needle, knife blade, or the "point" itself, until the *outer layer* of the skin is removed so as to show a *punctate moist surface*, free bleeding must be avoided, and the procedure is practically painless if properly performed. The vaccine is placed upon and gently rubbed into the denuded area, allowed to dry

Humanized lymph *passe.*

Bovine lymph.

Points and glycerinated lymph.

Vaccination technique.

* Dr. Hare quotes a case in which the father of a family in which smallpox had appeared, having been vaccinated in infancy refused further vaccination though exposed, at the same time permitting it in the case of his wife and three unaffected children. Of these one developed a mild varioloid and no others were affected at all but the father promptly developed a severe attack.

† Each maker supplies directions with his tubes.

thoroughly and afterwards the affected portion is covered with one of the numerous forms of "shields" readily obtained at any drug store.

Shields.

Sequence of Events in Vaccination.—A *reddish papule should make its appearance in from three to five days, promptly becoming vesicular, filled with clear lymph and later umbilicated. Its maximum development is reached at the end of* 7 *or* 10 *days when its contents become purulent in appearance.* A brilliant areola surrounds the pustule, underlain by a tender and more or less firm or brawny area. During the succeeding 3 or 4 days pain and tenderness become localized along the region of the lymphatics and their tributary glands may be slightly or decidedly swollen and inflamed, with fever and malaise.* The itching is intense, and scratching frequently causes secondary infection, hence in infants the movement of the arms must often be restrained. *Occasionally a general eruption closely resembling chickenpox may appear or there may be a distinct roseola.* In from 10 *days to two weeks* these symptoms disappear, *desiccation being complete as a rule by the end of the* 15*th or* 16*th* day, *the scab falling about a week later.* A depressed pitted scar is left behind varying in size with the severity of the process. All sorts of variations may be met with, such as long periods of incubation, entire absence of constitutional symptoms, or, in some instances, a more prolonged and severe stage of suppuration.

Duration of scar.

A thoroughly good vaccination usually leaves a scar which persists throughout the whole life of the individual. In an unvaccinated person, one should never rest content with a single vaccination, and in the presence of direct opportunity for infection or during an epidemic it is wise to repeat every vaccination if the first fails. It would appear certain, moreover, that cases presenting unusually severe symptoms owe these rather to secondary infection than to the true virus, so that some severe "takes" are less protective than the thoroughly typical and milder form.

"Pseudo takes."

The Previous Health.—Common sense should determine the propriety of vaccination in exceptionally delicate and diseased children, and a proper period chosen. So also as to the site of the vaccination the female infant or adult should, as a rule, be vaccinated on the leg'

Site of vaccination.

*The signs of inflammation and glandular abscess are often far more alarming than the actual result merits but, rarely, septic conditions are encountered. In an experience covering nearly two decades and including two years of municipal relief service the author has never had or seen one case of death, deformity, paralysis or invalidism and is convinced that the few bad results reported are almost invariably due to previously existing syphilitic or tuberculous taint.

the male on the arm, the resulting scar being of no moment to the latter.

VARICELLA.—(Chickenpox.) A trifling infection of unknown causation characterized by transient fever and a vesicular eruption.

Etiology.—Children are peculiarly susceptible between the ages of one and eight years, adults remarkably resistant. It prevails chiefly in the spring and autumn and spreads rapidly, is not inoculable, and one attack usually protects for life.

Symptoms.—In from one to two weeks after exposure the symptoms of mild infection appear. *Malaise, chilliness, pain in the legs and back are associated with a mild fever, rarely exceeding* 101–102°*F. Some children show no fever or signs of illness save the eruption which appears within* 24 *hours in the form of minute papules changing in a few hours to vesicles.* The face, scalp and neck are first attacked and it spreads to the extremities and trunk, being especially well marked in the back and attended by troublesome itching. The following points serve to distinguish varicella from smallpox or varioloid:—(a). *But few or no vesicles in a given case umbilicate.* (b). *The contents are serous, not purulent* though a few may suppurate and leave pits. (c). *The uniocular vesicles are emptied by a single* puncture. (d). *They rarely have an areola.* (e). They come in *successive crops* lasting two or three days *and form superficial crusts* which desquamate in a week or ten days. (f). *The vesicles therefore are seen in all stages*, i.e., *the eruption is not uniform.* (g). *The spots may be few in number, are scattered widely and do not tend to become confluent.* (h). *The secondary fever of suppuration is lacking.* (i). *Vesicles rarely appear upon the palms or soles.*

Trivial symptoms.

Vesicular eruption (2d day).

Successive crops.

Multiform.

Isolated.

Duration.—A week to two weeks. **Prognosis**.—Almost invariably good.

Complications.—Ordinarily lacking and seldom serious. The eruption occasionally appears in the mouth and hard palate, but rarely or never involves the conjunctivæ, larynx or trachea. The vesicles always variable in size, in rare instances become bullæ, and in any case the harmless vesicles may be infected by scratching. Cachectic children in this as in other infections may present a peculiarly virulent eruption.

Usually absent.

WHOOPING COUGH.—(Pertussis). **Definition**.—An infectious disease of childhood of unknown causation, self limited and immunity conferring, characterized by peculiarly violent paroxysms of cough, followed by a "whooping" sound. The disease is highly contagious, both before the appearance of and a week or two after the cessation of the

Highly contagious in all stages.

characteristic paroxysms but adults enjoy comparative immunity. It is a disease chiefly of early spring.

Incubation.—Two days to two weeks. **Duration.**—Eight to ten weeks. **Prognosis**.—Good in robust young children, bad in weaklings, nurslings and the aged.

Morbid Anatomy.—Practically none save that of a complicating diffuse bronchitis, broncho-pneumonia, emphysema or cardiac overstrain. Death by inanition, starvation and inhalation—or deglutition-broncho-pneumonias are rare phenomena.

Peculiar cough.

Symptoms.—*A coryza or mild bronchitis* is associated with a short barking *cough, progressively nervous and explosive* in character and tending to multiply in each paroxysm, the protracted expiratory efforts resulting in glottis spasm and a terminal long drawn stridulous inspiration, the "whoop." The seizures vary in severity and frequency,

"Whoop" may be absent.

absence of the whoop may obscure diagnosis, but the cough is peculiarly explosive and paroxysmal. Attacks may be almost constant or occur but once or twice a day.

Paroxysms easily excited.

Any emotion, pleasurable or otherwise, food, dust, over-heating or a cold draught may provoke the dreaded paroxysm. The child may vomit, the urine or fæces may be passed involuntarily, and hemorrhages may occur from the nose, ears, or under the skin or conjunctiva. The *blood* shows a leucocytosis, the lymphocytes being *relatively* increased. The *urine* may contain a trace of albumin and *fever* may be present or absent.

Depends upon cough.

Diagnosis.—*The presence of an epidemic, a persistent bronchitis, and the paroxysm of short, barking, rapidly repeated coughs with or without the whoop is sufficient for diagnosis.*

Complications.—There are many. It is frequently associated with measles, with subcutaneous, interstitial and true pulmonary emphysema,

Many and grave.

pneumo-thorax, persistent vomiting leading to inanition, enlargement of the bronchial glands, general convulsions, cerebral hemorrhage, broncho-pneumonia and lobar pneumonia, or even pulmonary tuber-

Adenoids.

culosis. *Adenoids* greatly increase its dangers and add to the severity of the paroxysms.

A formidable disease.

Comment.—*The laity regards* pertussis too lightly. *It is a disease of high mortality* through its pneumonic complications, often induced by a foolish disregard of ordinary precautions. Of children under one year, 25% die. Between one and two years 15%. Above these ages the mortality is slight, but it will be seen that the disease is formidable

Infant mortality high.

and deadly if lightly regarded. *In the public services of great cities it stands second to pneumonia as a cause of death in infants.*

SYPHILIS.—(*Lues venerea*, pox.) **Etiology.**—The *Spirochæta Pallida* of Schaudinn and Hoffman seems to be constant in active lesions and syphilitic blood but is not yet fully proven as the causative agent, and other organisms closely resembling, if not identical with it, are found in diverse lesions and situations in non-syphilitic cases.

Germ unproven.

Mode of Conveyance.—Syphilis in an active form may be, and usually is acquired, less often inherited. Dilute inherited syphilitic infection is of course well nigh universal but is symptomless. It is both contagious and infectious, confers almost absolute life-long immunity and furthermore *is capable of affecting each and every structure of the human body so diversely as to make it the chief of protean diseases.* Given an abraded skin or mucous membrane in a non-immune and contact with the secretions or blood of a syphilitic and infection almost certainly follows. Less frequently prolonged contact or retained virus alone may produce it on a sound mucous membrane. The disease is essentially venereal, sexual intercourse being the means of transmission and sexual organs the usual seat of primary lesions. Yet owing to the irregular and unusual forms of intercourse and the communicability of the disease through certain secondary lesions and by means of intermediate substances, we find labial, lingual, tonsillar, mammillary and rectal infections as well as the digital sore not infrequent in physicians, dentists, laundresses, rag pickers, etc. It follows also that syphilis may be innocently acquired with or without sexual congress. Husbands infect innocent wives and less often a guilty wife transfers the disease of her lover to an unsuspecting husband. The innocent girl carrying an oral chancre for which the mucous patches of her fiance are responsible is no rare client and may infect other family members. *The individual syphilitic is a menace to all about him during the active stages, as the secretions of his lesions, his blood and his lymph, are alike virulent.* Fortunately breast milk, saliva, and usually the semen, are not infectious unless contaminated through local sources. The late lesions (tertiary) are seldom sources of infection unless relatively early or co-existent with late secondary lesions. High degrees of heat or cold seem to destroy the virulence of the secretion otherwise active even in a state of desiccation.

Readily transmitted.

Protean.

Essentially venereal.

Occasional variations in site.

The syphilitic a menace.

Tertiary lesions not often contagious.

Modifying Influences.—As to its later course no correct inferences can be drawn from the primary symptoms or the site of the lesion. though *the general rule governing all infections which leads us to expect the severer manifestations in persons of bad habits, poor physique or a vitality impaired* by *antecedent or co-existent chronic disease holds good*

As in other ailments.

Acquired lues in children.

in this one. The ravages of *acquired* syphilis in children are exceptionally violent and widespread.

The Three Stages.—*The primary stage of syphilis comprises the time elapsing between the recognition of a local lesion at the seat of infection and the appearance of the constitutional symptoms and eruption of the secondary period.* It is represented by the development of the chancre and coincident swelling of the adjacent lymph glands (syphilitic buboes).

The third stage or tertiary period marked by soft nodular growths of peculiar structure (gummata) may be entirely lacking in efficiently treated cases or may appear despite the most active and prolonged specific medication. Extending beyond these circumscribed and classified periods is an *insidious toxæmia of indefinite or life-long duration* which makes

After effects.

the individual liable to develop degenerative diseases of an apparently non-syphilitic nature. Such are locomotor ataxia, paretic dementia, arterio-sclerosis, apoplexies, aneurism and the nephritides. This fact has led life insurance companies to exact a long period of immunity and evidence of radical and efficient treatment as perquisite to acceptance

Insurance experience.

as "first class risks," yet the actuarial report of the "combined investigation" committee shows the futility of the requirement by the enormously excessive death rate experienced.

The Initial Lesion or Chancre and the Bullet Bubo.—After an

Incubation.

incubation period of from 10 days to 6 weeks the first characteristic

Papule or erosion.

symptom is apparent in a *single papule* often a part of an *herpetic eruption* at the *point of infection* whether genital or remote which increases in size, becomes *indurated* and may remain as a dry plaque or show an eroded surface with scant secretion.* The feel is that of a

Induration.

split pea save that in certain cases especially in the female the induration may be lacking. Its usual location is the vulva or the prepuce, but it may appear upon any portion of the genitals or in neighboring or distant parts *and always represents the actual site of infection.* The glands representing the lymph drainage of the affected part undergo a painless

Bubo.

enlargement, known, when in the groin, as the *bullet bubo.*

The Secondary Stage.—*Within from 6–8 weeks after infection systemic infection is manifested by general glandular swelling, mental*

Constitutional symptoms.

depression or irritability, insomnia, malaise, pallor, pain on pressure over or near the sternochondral joints, neuralgia, nocturnal headache, joint

* Multiple lesions are not uncommon in women but are relatively rare in men, and sometimes "satellite" indurations of later formation may be observed.

pains and exertion-dyspnœa, fever being rare or if present, trivial. *As these subside the exanthem develops.* Exanthem.

Exanthemata of the Secondary Period.—Their characteristics must be thoroughly understood because of their great importance in diagnosis. These are:—(a). *Their polymorphous tendency.* This may merely represent different stages in the development of a certain type or show a true mixed eruption. *Such polymorphism is the rule in syphilis.* (b). *Symmetry and tendency to general distribution.* (c). *The tendency to follow skin lines in arrangement.* As is seen especially in profuse eruptions and particularly in the oblique parallel lines of eruption from the back downward obliquely to the front. (d). *The absence or trivial degree of itching.* (e). *The tendency to oval illiptical or circular forms.* (f). *The prompt response to mercurial inunctions.* (g). *The tendency to shift their pigment to the periphery of the lesion** is characteristic especially in the case of disappearing or past lesions. In the former a central faint red may be surrounded by a lighter area, in its turn encircled by a deeply pigmented border. In the latter the whole inner area is white (syphilitic leukoplasia). Peculiarities *must* be known.

Varieties of Syphilitic Exanthemata.—*The earlier syphilitic eruptions show a marked tendency to predominant involvement of the abdomen, front of chest and anterior surface of arms.* Early eruptions.

Roseola.—This earliest form *chiefly affects the trunk and is so evanescent* and otherwise symptomless as, in many instances to escape the patient's notice.† The *brownish red pea sized macules* are seldom raised above skin level and leave no trace. Earliest form often overlooked.

Large Macular Syphilide.—Unlike roseola this is due to actual round cell infiltration, *is usually slightly raised* and *frequently, by central fading or border coalescence, forms the varieties known as "gyrate," "circinate," "annular," etc.* The *color* is *livid* in the lower extremities, *copper color* in the upper, and its pigmentation may persist for considerable periods or give place to the white areas known as cutaneous leukoplasia. Readily recognized.

The Papular Syphilide.—*This may be pure or mixed with the macular or pustular form and is the commonest syphilide and the most diverse in size and form.* It may be distinctly papular, nodular or flattened, and in size varies from a pin head to 2 cm. or more. *Its distribution may* Commonest type.

*The *coppery or ham colored eruptions* though suggestive are neither invariable in nor are they peculiar to syphilis.

† Dashing ether upon the surface of the body will often bring out a phantom like but clearly specific roseola.

Distribution.

*be general or in later types limited to the genitals, palms, soles or other areas.** Resembling psoriasis in some respects it lacks the silvery scales and peculiar localization of that disease and seldom involves

Lenticular.

large areas, occurring usually in small patches. The *lenticular form* is a rash of brief (7–14 days) duration followed by desquamation and leaving no permanent markings. Its nodules are grouped and especially involve the extremities and trunk. The flat glistening form involves especially the face and is *sharply defined, flattened and centrally depressed. It* also leaves no permanent marks. The *lichenoid* papules are *grouped, not generally distributed, persistent, become crusted and leave tiny pits.* Among the later forms are the *orbicular papules* which show a *ring shaped border and central depression,* and chiefly affect the genital

Lichenoid.

Tubercular.

region. The *tubercular form,* very *late* and *persistent and closely allied to tertiary eruptions,* is *grouped, scaly or crusted, richly pigmented, and may terminate in ulceration which leaves permanent scars.*

The Pustular Syphilides.—These are *rarely early but may co-exist with the papular form and are associated with marked general symptoms* (fever, malaise, pallor, etc.). *The exanthem may closely resemble acne*

Suggests variola.

or even smallpox at the outset, and *may form extensive crust formation. Ulceration, superficial and spreading* (echthyma syphilitica) *or deeper,* may occur and the crusts may so arrange themselves as to be termed syphilitic rupia. *They leave peripherally pigmented smooth shiny loose scars. Hemorrhagic forms* of syphilides occur and mean a serious

Hemorrhagic types.

case as in other exanthematous diseases.

Palmar Syphilides.—Both the palms and soles may be involved in the late secondary papular syphilides and *this eruption is of almost pathognomonic significance.*† If these papules form between the fingers or toes the result is maceration and oftentimes decided inflammation and if the matrix of the nail or its margin be attacked *syphilitic onychia*

Onychia and perionychia.

is produced, the nail becoming dead and being finally cast off. There is frequently a *perionychia* due to suppuration at the nail margin.

Excoriations.

Indurative Edema.—On and near the genitals the secondary processes may assume a most severe type of *excoriation, inflammation and even indurative edema* and *these lesions are highly contagious* and probably the most frequent source of infection. *Fissures, ulcers* and *venereal papillomata* are associated with this condition.

Fissures, ulcers and warts.

*Any papular or pustular form may be hemorrhagic in grave or complicated cases.

†The late Mr. Berkeley Hill used to say that he always taught his *students* that any eruption in the palm *was syphilis,* and the exceptions are so few that the dogmatic statement was justifiable.

The Hair.—Syphilis of the scalp commonly takes the form of *pustules* which by coalescence may produce *more or less irregular or scattered spots of baldness*, sometimes replaced by gray hair, but usually, under efficient treatment, promptly taking on a normal new growth. A less common form is the *seborrhœic which leads to diffuse baldness.* Baldness.

The Buccal Cavity.—This region is one of the most important in connection with the diagnosis of syphilis in its secondary stage. The earliest change in the tongue consists of *prominence of the papillæ with small spots of whitish exudate;* these areas later becoming raw and glistening, and, at the edge, converted into ulcers. Many variations occur in the form and distribution of such lesions. *The faucial pillars, tonsils and soft palate should always be inspected as well as the lips and cheeks where fissures and ulcers may be detected. Indeed the kidney shaped, gray bordered ulcer of the tonsil is one of the earliest secondary manifestations and may be painless and so escape the patient's notice.* Tongue. "Mucous patches." Important regions.

Tertiary Syphilis.—*This is essentially the gummatous stage and should never appear in a marked form in those of good constitution who have received adequate treatment,* though in those less fortunate it assumes the most terrible types of destructive lesions, *affecting the osseous as well as the soft tissues* and leading to frightful suffering and humiliating deformity. *Large pustules* may appear in this stage (pustula major) with rapid necrosis and coalescence, foul discharge and severe pain, or the various *gummatous neoplasms* may develop in various parts of the body, *internal and external.* The *cutaneous gumma* varies in size from a buck shot to a mandarin orange. *It is somewhat soft in consistency and at first spares the true skin; the tendency, however, is to disintegration which may or may not involve the skin according to the deep or superficial situation or the growth.* In certain instances the peculiar *serpigenous ulceration* so characteristic of the disease may appear and leave its characteristic *sinuous, reniform* or *semicircular scars.* The gummata may involve the *bone, periosteum, muscles, joints or tendons, the painful swellings of which are common in the earlier stages* and if the inflammation be severe may result in fibrous anchylosis. May never appear. May cause destructive lesions. Cutaneous. Serpigenous ulceration. Osseous forms.

Syphilis of the stomach has already been referred to, *the soft palate is often affected* at its junction with the bony portion, the process tending to *a pathognomonic perforation* (see page 7); or, the *hard palate* itself may be involved. Indeed *space will not permit a description of the gummatous changes, inasmuch as they may involve any region of the body and are dealt with in other sections.* Soft and hard palate.

The Lymph Glands.—*A general glandular hyperplasia characterizes*

the secondary stage and may be exaggerated in any group of glands which drains an ulcerating surface. *The post cervical, inguinal, axillary, epicondylar and posterior mastoid glands* are especially affected and are easily palpated. Occasionally a similar glandular swelling occurs in a tertiary period, not infrequently ending in necrosis. *The tongue* may be the seat of tertiary as well as secondary changes in the form of *leukoplasia or gummata*, the latter of which may produce painful or permanent *fissures and deforming scars*, as the result of ulceration. The so-called *syphilitic psoriasis* as affecting either the tongue or buccal mucous membrane is recognized by the presence of *whitish patches which are hard and horny* to the touch. *These are easily eroded and constitute an intractable and painful tertiary lesion.*

Glands most accessible.

Tongue.

Buccal membrane.

Hereditary Syphilis.—Little need be said concerning the appearances in this condition beyond that contained in the first section of the book. The unfortunate inheritors suffer especially from the following lesions: (a). *Syphilitic hemorrhage of the newborn* leading to death shortly after delivery. (b). *Ulceration and hemorrhage from the navel* usually uncontrollable and resulting in death. (c). *Snuffles.* The nose is blocked by syphilitic inflammation and there is usually an *excoriating secretion* and a *specific periostitis which may produce saddle nose.* (d). *Excoriations, due to maceration of papular and pustular* eruptions, *about the nates.* (e). *Marked nutritive disturbances* which give to the child a *wizened, senile appearance*, the skin being dull, inelastic and often hanging in folds. (f). *Pemphigoid lesions*, which occur early in infancy (the first few days) and *usually justify a fatal prognosis.* In addition one finds enlargement *of the liver and spleen, epiphyseal inflammation and even separation, iritis, keratitis, conjunctivitis, etc.*

Pathognomonic signs.

"Snuffles."

"Sore bottom."

Senile physiognomy.

Syphilis Hereditaria Tarda.—This term is applied to *those forms of hereditary syphilis which appear about the fifth year or sometimes as late as the twelfth and tend to last until the age of twenty or twenty-one.* The *Hutchinsonian syndrome* is:—hazy cornea or actual interstitial keratitis, increasing deafness and crescentically notched, prominent upper incisors. To these Mracek would add a flat or depressed nose, fine scars at the angle of the mouth, on the upper lip, or on the mucous membrane of the lips *radiating* from the nares, and prominent frontal bosses, together with a manifest arrest of development.

Jonathan Hutchinson's syndrome.

Comment.—The student must remember that *syphilis is the most protean of diseases and frequently assumes forms unrecognizable by any save therapeutic tests.* Whether the patient be infant or adult, male or female, cleric or convict, as a possibility it always exists, for the

Therapeutic test.

innocent suffer even as the guilty though less often, and the physical results of the errors of youth cannot always be obliterated. In this condition it may be said that *when one encounters conditions peculiarly baffling and indeterminate* in their symptomatology, *syphilis* and *drug addiction* should always be considered.

No one above suspicion.

Syphilis and drug addiction.

ERYSIPELAS.—Definition.—An acute infectious and contagious disease due to the streptococcus pyogenes and characterized by a spreading dermatitis with marked toxæmia. **Etiology.**—The disease is particularly common and easily induced in women after delivery, cases of surgical operation and in elderly patients or those debilitated by chronic disease. Syphilitics are especially liable and may suffer excessively.*

Once common, now relatively rare.

Syphilitics.

Symptoms.—After an *incubation period* varying from three days to a week and with or without *slight malaise, tingling or burning* of the affected surface, there is *chilliness or an actual rigor* and a *high, somewhat irregular fever* (103–105 F.) associated with *marked leucocytosis. The affected skin is dull crimson, swollen, tense, persents a slightly raised edge* and later becomes covered with blebs. In favorable cases it remains but a few days, gradually fades and is followed by desquamation, the fever ending usually by crisis. *Relapses are common* and in unfavorable cases the process may shift its seat and a typhoid state may develop. **Differential Diagnosis.**—The author knows of no other disease that presents these symptoms.

Pathognomonic signs.

PYÆMIA AND SEPTICÆMIA.—These are essentially surgical conditions and need no extended description in this volume.

Diagnosis.—This depends upon a *septic temperature or one which may resemble at first that of typhoid fever* or malaria but is associated with *marked leucocytosis* in any resistant patient, *lacks the Widal* test *of typhoid and hæmamœba of malaria* and is oftentimes associated with scarlatina-like rashes or toxæmic jaundice. *Pyæmia* must be traced to its *focus* and *is distinctly septic from the start* having a widely *remittent or intermittent* temperature associated with *recurring chills and sweats.* Aside from the surgical conditions these two diseases are likely to be confused only with typhoid fever in the former case and malaria in the latter or with both. Ordinarily care and a study of the symptoms is always sufficient to make a differential diagnosis. It is also necessary, however, to have in mind *malignant endocarditis* as a

Differentiation.

Typhoid and malaria.

Surgical conditions sequelæ.

* In one case of imperfectly treated syphilis the onset of erysipelas was attended by *immediate* periostitis and necrosis of the facial bones which produced tremendous deformity and death within a few days.

Tuberculosis.

possibility, and the heart should be examined in every case where either septicæmic or pyæmic symptoms are present. Advanced tuberculosis is a septicæmia in part but offers few difficulties in its pulmonary form, and as an acute miliary process lacks leucocytosis.

A vanishing disease.

LEPROSY.—*This, the most ancient of known diseases, is caused by the bacillus lepræ** and still prevails throughout the world, though to a very limited and rapidly diminishing degree in Europe and North America, and, indeed, in all countries where modern methods of control through segregation have been attempted.

Slightly infectious.

Mode of Conveyance.—Much difference of opinion still exists concerning the direct and hereditary conveyance of this disease. As regards the latter it is probable that leprosy, like tuberculosis, may, but rarely does exist at birth, and that it is in nearly all cases acquired, the young children of leprous parents being especially exposed to the bacilli which are found in nearly all excretions and secretions of affected cases. Most intimate contact seems indispensable to infection, inasmuch as nurses and doctors in leprous settlements are rarely attacked.

Suggestive resemblance.

Development of Leprosy.—The close resemblance of leprosy to tuberculosis in many particulars is no less remarkable than the likeness of its stages to the development of syphilis. There is of course an initial lesion believed by many to be represented by an ulcer on the nasal septum. There is a *prodromal stage* resembing the second stage of syphilis in that it is associated with transient fever, headache, joint pains, and commonly with epistaxis, possibly due to the *septal ulceration*, and exhausting sweats may occur without fever. After several months or even a year or two a febrile paroxysm is accompanied by *macular eruptions*. The circumscribed erythematous patches are generally distributed, chiefly affecting the back of the hand, the forearm, the face, the malar and supraorbital regions being especially affected, the scalp usually spared.

Primary sore and constitutional symptoms.

Macular eruption.

Macular anæsthesia.

"The silver men."

Third stage.

The macules may be *hyperæsthetic* early, but later become *anæsthetic*, either centrally or wholly. They are at first transient but tend to become *persistent and pigmented*. Without further advance they may show a loss of pigment and then constitute the *dead white, shiny* areas of "white leprosy." A definite *third stage* is represented by three forms, the *nodular or tubercular*, the *anæsthetic, and the mixed*.

*The bacillus is morphologically indistinguishable from the tubercle bacillus and resists decolorization in much the same manner. It cannot be readily cultivated; however, stains much more easily and does not produce tuberculosis in inoculated guinea pigs.

Nodular leprosy may lack the macular stage and the *subcutaneous tubercles are flat, hard and elastic* to the touch, and *most numerous about the ears and face.* Their increase in size and the infiltration of the affected parts produce the "*leontine facies.*" With occasional disappearance and reappearance in individual areas, they nevertheless multiply and ultimately involve the greater portion of the body and mucous membranes and break down into ulcers which cause horrible deformities from tissue loss and cicatricial contraction.

Leontine facies.

Necrosis and deformity.

Anæsthetic Leprosy.—This is characterized by an extensive painful neuritis due to direct invasion of the nerves by the bacilli. *Extensive* anæsthesia results, associated with bullæ, spreading ulceration, and subsequent contractures. Muscular atrophy is widespread, and *fingers and toes may be completely destroyed.*

Mixed Leprosy.—Represents merely a combination of the two forms.

Differential Diagnosis.—Any difficulty is limited to the early cases of the macular or anæsthetic type. Three diagnostic criteria are available. (1). *The easy recognition of the bacillus in the blood of a leprous nodule, or, in anæsthetic cases, in the fragment of a nerve, usually also in the nasal secretion which may be increased by the administration of an iodide.* (2). *The central anæsthesia of early macular areas.* (3). *The administration of pilocarpine to show the absence of perspiration in the involved areas* (Baelz).

Three diagnostic criteria.

ANTHRAX.—(Malignant pustule, charbon, wool sorter's disease). This world-wide scourge of cattle and sheep may be conveyed to man through direct contact or by the handling of wool and hides, the channels of entrance being chiefly wounds or abrasions of the skin, the pulmonary tract, the infected stings of insects and the gastro-intestinal tract. The germ is strikingly resistant, the spores especially so, infection being possible after years through laboratory cultures or in pastures containing the buried bodies of infected animals. It grows readily on ordinary media at temperatures between 12° and 45° C., producing in gelatine stab cultures the inverted pine tree figure. They produce spores only in culture and stain with all ordinary dyes. The disease is rare in the United States, very frequent in certain parts of Russia, particularly Siberia.

Sources of infection.

Fig. 171.—Anthrax bacillus (bacillus anthracis). A non-motile, non-chromogenic, sporogenous aërobic bacillus, readily cultivated, stained by all methods and highly resistant.

Vitality of spores.

Symptoms.—The disease may be external or internal, in the

former assuming the type of "malignant pustule" or "malignant anthrax edema." The latter form is usually pulmonary, more rarely intestinal.

External forms.

Malignant Pustule.—From three days to a week after exposure the development of a small itching papule is followed by a rapidly increasing inflammation and infiltration and the formation of a hemorrhagic vesicle which becomes necrotic and bears upon its infiltrated marginal zone similar lesions. Coincident with the rapid spread of the inflammation adjacent lymphangitis and glandular swelling are evident. The induration and edema are extreme and the necrosis may be extensive yet pain is slight or absent. Marked constitutional symptoms are evident in 48 hours when the germ reaches the blood but the active septic phenomena *including the fever* subside in the later stages, the septic typhoid state preceding death.

Characteristic lesion.

Constitutional symptoms.

Curious predilection.

Malignant Anthrax Edema.—Primary constitutional symptoms, excessive gangrenous edema with a predilection for the eyelids, head, hand and arm characterize this variety; the patient's mind may be clear and free from apprehension in either form.

Pulmonary, gastro-intestinal and cerebral symptoms.

Internal Anthrax.—(a). *Wool sorter's disease.* A sudden onset with chill, prostration, severe pain in the back and extremities, a temperature (102–103) with chest pain, rapid breathing and in some instances marked bronchial symptoms, characterize this form. The pulse is weak, vomiting and diarrhœa may occur, violent cerebral symptoms are noted and death may result within 24 hours. (b). The *intestinal form* is due to ingestion of the germs and is characterized by chills, moderate fever, vomiting, diarrhœa, marked prostration, dyspnœa, phlegmon or petechiæ and enlarged spleen. Hemorrhage may occur from free mucous surfaces, and unlike the external form, anxiety is marked and there may be terminal convulsions.

Hemorrhage.

Occupation.

Diagnosis.—The occupation of the individual is almost always suggestive and the history of an itching papule and its vesication and necrosis should lead to an immediate examination of the contained serum, either microscopically or by inoculation of white mice which are very susceptible. A progressive course with *falling fever* eliminates phlegmonous erysipelas and diffuse cellulitis and malignant edema shows tissue crepitation. The germ can often be recovered from the blood within 48 or 72 hours after the onset, but may be long delayed.

Falling temperature.

Prognosis.—The mortality is variable but always high in the internal forms and those of the external type in which the face is involved; less so if the extremities are affected.

HYDROPHOBIA.—(Rabies, Lyssa.) A fuller knowledge and more radical precautions have rendered rabies a rare disease in America, England and Germany. It depends upon a specific virus conveyed to man chiefly by the dog, though most animals are susceptible in varying degrees, as is proven by successful inoculation and by cases observed in cats, skunks, wolves and even cows. *The salivary secretion is the chief carrier,* the *nervous system the main focus* of this unknown poison. The incubation period varies inversely with the age of the victim, is shortest when following wounds of the face and head, but depends also upon the size of the wound, the protection of clothing and the animal conveying it. The infected dog stands below the wolf and cat in virulence. It is stated that infection follows in about 15% of dog bites as compared with about 40% for wolves but it would seem that the severity of the lesion might be chiefly accountable. The mortality varies from 50 to 80%, but has been greaty reduced by the Pasteur treatment.

Saliva the medium.

Important factors.

Symptoms.—*The Premonitory Stage.* This is characterized by irritability, insomnia and apprehension and marked mental depression, photophobia and hyperacusia may be present. Hoarseness and dysphagia usher in the second stage.

Period of Hyperæsthesia and Spasm.—(Duration 2 or 3 days). The slightest stimulus, physical or mental, excites painful and violent reflex spasms affecting chiefly the mouth and larynx and accompanied by subjective dyspnœa. The mind is clear except during the convulsions and the paroxysms excited by attempts to swallow even water account for the use of the term "*hydrophobia.*" Fever may be absent but is usually present in moderate degree. Attempts to injure others are rare and confined to the paroxysms.

Extreme irritability and spasms.

Terminal and Paralytic Stage.—Subsidence of the spasms, gradually developing coma and progressive cardiac weakness terminate life.

Lyssophobia.—(Pseudo-hydrophobia). This is merely an hysterical manifestation on the part of one bitten by a suspected animal, fever is rare and the signs of hysteria usually manifest, though naturally the irritability, apprehension and mental depression may exactly simulate the premonitory stage of the true disease. The excessive hyperæsthesia, true spasm and other indications of actual infection can hardly be exactly simulated.

Imaginary hydrophobia.

Diagnosis.—*The greatest enemy of the patient and diagnostician is the officious public servant or citizen who promptly destroys and disposes of a suspected animal,* for the first step should be the transfer of the living

Fool heroes.

animal or of the fresh brain and medulla to the hands of the bacteriologist the purpose of the inoculation of animals and the identification of the Negri bodies.* Hence if possible the canine suspect should be kept confined and carefully watched, and if killed be transferred at once to competent hands. This should seldom be necessary for actual diagnosis as the clinical picture is clear, but it often prevents an unreasoning and unfounded dread in a community where some halting physician diagnoses hysteria as rabies and causes the persecution of a wholly innocent dog population.

Diagnosis usually easy.

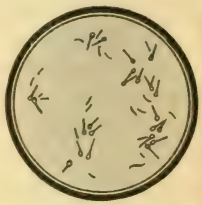


Fig. 172.—Tetanus bacillus (Bacillus tetani). A long slender bacillus with single polar spore, flagellate, motile, anaërobic, non-chromogenic, sporogenous, liquefying, readily stained by all methods but cultivated with difficulty.

TETANUS.—(Lockjaw). The bacillus responsible for the lockjaw is an anaerobic germ, motile, inoculable in animals and yielding an antitoxin which permits the production of a protective serum. Its peculiar toxin is fatal in the extraordinary dose of 0.23 milligram. As *tetanus neonatorum* the disease especially affects newborn children in countries or districts where uncleanly methods are followed in the care of the navel. The soil of certain regions is peculiarly infectious and the germ usually enters the body through a *punctured* or *contused* wound. The fatalities attending the use of toy pistols are well known.

Marvelous toxicity.

A soil dweller.

Incubation period.

Symptoms.—After a period of incubation of about a week or ten days, or more rarely two weeks, a subjective sensation of constriction of the neck and jaws with difficult mastication is followed by a tonic spasm of the muscles which locks the jaw, the outdrawing of the mouth and raising of the eyebrows causing the *risus sardonicus*. By gradual involvement the muscles of the trunk and extremities are affected, producing rigid extension (orthotonos), or violent posterior flexion of the spine (opisthotonos), and more rarely lateral flexion (pleurosthotonos) or anterior flexion (emprosthotonos). The duration of the spasms is variable, relaxation usually incomplete; they are initiated by the slightest irritation and are extremely painful. The fever is usually moderate or perhaps absent, more rarely there is hyperpyrexia. Spasm of the glottis may cause dysphagia and paralysis of the facial muscles may

Locked jaw.

Risus Sardonicus.

Opisthotonos.

Fever.

* Negri's bodies are protozoon, angular, round or oval, varying from 1 to 20μ in diameter. Negri's original claims have been largely substantiated by other observers and the micro-organism seems to be almost or quite constant and found in no other disease. They are found most readily in the Cornu Ammonis. By this means a diagnosis can be made within 24 hours, and fortunately the bodies are quite resistant to post mortem changes (Poor).

occur, but in children spasm may be limited to the production of the risus sardonicus.

Diagnosis.—Strychnine poisoning lacks the early involvement of the muscles of mastication and the rigidity between spasms. (See p. 640.)

Prognosis.—Of idiopathic cases about 50% die, traumatic cases 80%. The head tetanus due to wounds of that part, is in its acute form, almost invariably fatal; if chronic, the mortality may only be 25%.

Head tetanus.

GLANDERS.—(Farcy). Primarily a disease of the horse, it is communicable to man through the agency of the bacillus mallei. It is essentially an infective granuloma, actual contact being the usual method of conveyance, and it is encountered in both acute and chronic forms.

Acute Glanders.—(Infection through the nostrils). Three or four days after infection there is fever and evidence of local sepsis. The mucous membrane of the nose becomes involved within 48 or 72 hours. nodules forming which undergo necrosis, the ulcers yielding a muco-purulent discharge. Pustules form about the face and joints, the cervical lymphatic glands are especially enlarged, the nose is swollen and pneumonia is a frequent complication. The disease is fatal in from 8 to 10 days.

Primary intra-nasal lesion.

Acute Farcy.—(Infection by the skin). The symptoms are those of an acute septicæmia following local phlegmon representing the site of inoculation, together with an acute lymphangitis characterized by subcutaneous nodules in the course of the lymphatics, which may suppurate and are called "farcy buds." The urine may show the germs. Death usually ensues in from 10 to 15 days. Both varieties of the disease show a chronic form lasting for months or even years and occasionally terminating in recovery. The inoculation of animals and cultural methods are often necessary for diagnosis. In all forms the occupation serves as a valuable suggestion and it is usually possible to find the source of infection in a diseased horse.

Dermal lesion.

"Farcy buds."

Chronic glanders.

ACTINOMYCOSIS.—This disease which also belongs to the infective granulomata is caused by the streptothrix actinomyces or ray-fungus, and constitutes the "lumpy jaw" of cattle. Successful cultivation and animal inoculation have proven the germ, and the pus from the lesion shows characteristic opaque yellow granules from $\frac{1}{2}$ to 2 mm. in diameter. Infection occurs, doubtless, through the entrance of the germs into the mouth or air passages of man, and the disease is divisible into four forms according to the seat of the primary process.

"Lumpy jaw."

Characteristic yellow granules.

If the *digestive tract* be involved there is swelling of the face, usually unilateral but sometimes bilateral, closely simulating sarcoma or tertiary syphilis. The tongue may be affected or the disease may be found along the digestive tract where it is reported to have produced specific appendicitis, colitis and even peritonitis but the liver is rarely affected.

Simulates pulmonary tuberculous.

Pulmonary actinomycosis is characterized by a chronic, wasting febrile disease with marked pulmonary symptoms simulating chronic bronchitis, disseminated tuberculosis, broncho-pneumonia, or tuberculosis associated with interstitial changes and cavities. In the digestive form the organism may be recovered from the stools, in the pulmonary form the presence of actinomycoses in the pus and the absence of tubercle bacilli makes the diagnosis clear.

Cutaneous actinomycosis closely simulates tuberculosis of the skin. *Cerebral actinomycosis* yields symptoms of brain tumor or abscess.

"Sunstroke" a misnomer.

THE INTOXICATIONS.—(*Sunstroke, thermic fever, insolation, heat exhaustion, siriasis.*) Great heat with decided humidity are the chief factors in both heat exhaustion and sunstroke. Mere exposure to the sun does not cause it, the highest degrees of heat being borne in the western deserts with less liability to sunstroke than would be experienced at a much lower temperature on the seaboard or in a marshy district. Furthermore, it occurs both night and day, in doors and out of doors.

Physical condition.

Physical exhaustion, lack of food or water, an unsound heart or kidneys, and the use of alcoholics are important contributive factors.

SUNSTROKE.—Symptoms.—The assumption that inadequate heat dissipation is associated with excessive heat production due to exhaustion of inhibitory centres, and the fact that at autopsy an extensive pulmonary, cerebral, hepatic, and renal congestion is present suffices to explain the symptoms. These are primarily vertigo and oppression followed by sudden coma with or without a convulsion.

Rational explanation.

Coma.

The facies is that of apoplexy *without paralysis*, i.e. lividity and cyanosis, stertorous breathing and pupils fixed in contraction or dilatation and there is an associated hyperpyrexia (105–112+). Death may occur within 12–48 hours. Relapses are not infrequent and the stroke may be followed later by meningitis. The after affects endure for years in the shape of discomfort on exposure to unusual heat and humidity or actual attacks of heat exhaustion or sunstroke. This acquired vulnerability is recognized by all life insurance companies.

Hyperpyrexia.

After effects.

Differential Diagnosis.—The extreme high temperature excludes everything save pontine hemorrhage which is itself excluded by the absence of paralysis.

HEAT EXHAUSTION.—This is characterized by subnormal temperature, sweating and collapse, and is wholly unlike sunstroke. Unlike sunstroke.

ALCOHOLISM.—The well known effects of large potations need not be described, yet many mistakes arise in connection with the unconscious or stuporous stage. It should be remembered that in alcoholism the pupils are usually dilated, the individual can be partially roused by pinching the inner side of the upper arm or thigh, that the temperature is normal or more frequently subnormal, the breathing, deep, slow but rarely stertorous. The fact that the breath is alcoholic is important but far from conclusive evidence. Acute.

CHRONIC ALCOHOLISM.—The symptoms of this condition may be patent to any layman, or entirely absent. The face of the sot requires no description here, but quite as great destruction may be going on in the tissues of one of his fellows whose habits may be wholly unsuspected, even, as happens in rare instances, by his family members. A certain spree drinker of the author's acquaintance has for years left home at regular intervals, gone to a hotel, quietly passed into an alcoholic stupor only to arise in the late forenoon of the following day, take a Turkish bath and proceed with the business of life. Such is the sneak drinker. Others drink openly and boisterously, yet others quietly, continuously and in a routine way without themselves suspecting the formation of any habit or the creation of secondary diseases. The kind of stimulant taken is important; pure beer and good wines are the least injurious, absinthe, brandy, whiskey and gin the most harmful. The poisonous sophisticated liquor so generally sold in low groggeries is peculiarly pernicious in its effects, and the drinking of strong or mixed liquor on an empty stomach so common in this country is an unmixed curse, fostered by the "treating" habit. Sneak drinkers. Unconscious drunkards. Sophisticated liquors.

Visceral Alterations.—*Digestive System.* Chronic gastric catarrh frequently associated with hepatic cirrhosis is a common sequence. Gastric dilatation in beer drinkers, and the coated tongue and foul breath of the heavy drinker of stronger liquors is well known. *In the kidneys* the chief change is found in simple hypertrophy though interstitial nephritis is undoubtedly promoted by the drinking habit. *The lungs* seem to be rendered more vulnerable to bronchitis and tuberculous processes, the circulatory system shows a tendency to myocardial degeneration and arterio-sclerosis. *In the nervous system* both general and local changes may occur, the morning depression, mental irritability, impaired concentration and quickness of perception, failure of memory and a decided change in the moral character being common Cirrhosis, gastric dilatation. Renal and circulatory lesions. May simulate "paresis."

and well known manifestions. Recurrent or persistent tremor, especially marked in the morning hours, and even transient or persistent delusions may occur.

Alcoholic neuritis is described on page 606. Alcoholic epilepsy is a curable form occasionally encountered and the so-called "wet brain" and its symptoms have been described on page 610. One of the unfortunate results of the early morning malaise, impairment of digestion and mental depression is the fact that it largely or wholly disappears if a brisk stimulant is taken and thus the vicious cycle is maintained.

"Morning bracers."

DELIRIUM TREMENS.—(Mania a potu.) This condition is invariably associated with chronic alcoholism, never with a spree of whatever length in an habitually temperate person. In the chronic drinker it may result either from some excess or from sudden withdrawal and is frequent in such persons following an accident, surgical operation or some great emotional shock. Its association with acute disease and especially with pneumonia is well known to clinicians.

Results from *chronic* alcoholism.

Symptoms.—One of the first evidences is tremor associated with a marked depression, restlessness and insomnia, which leads to increased potations. Within 24 or 48 hours or even less an active voluble delirium appears and sooner or later the well known hallucinations known vulgarly as "the horrors." Friends become enemies seeking to deprive him of life and liberty; the commonest noises are misinterpreted; imaginary snakes, rats or other animals surround him, and he is in a constant and pitiable state of terror. Such patients need to be watched, and, usually, restrained, though seldom dangerous save to the furniture and themselves. The tremor of the tongue and hands is extremely marked, there may be a fever of mild degree, insomnia is constant and the disease subsides by lysis in three or four days unless death occurs from exhaustion and heart failure. The frequent existence of some serious injury and the development of pneumonia in these cases should never be forgotten. This is particularly true of those patients seen in public services, who have been brought in by the police. Indeed apical pneumonias are not infrequently associated with a delirium closely simulating that of alcoholism. The fact that 10% or more in such services die, emphasizes the importance of the ailment. Recurrence follows continued excessive drinking and usually shows an increased severity and higher mortality.

Tremor, insomnia, depression.

Delirium.

Hallucinations.

Restraint required.

Mode of death.

A common error.

Recurrences.

MORPHINE AND COCAINE HABITS.—These topics have been discussed under case-taking, see page 40.

CHRONIC LEAD POISONING.—The relation of occupation to

lead poisoning has already been discussed, see page 44. Accidental contamination of drinking water by lead is common through faulty plumbing, the use of cosmetics and hair dyes and the adulteration or faulty preparation of foods and beverages. It is claimed that females are more susceptible than males, and adults more than children.

Symptomatology.—The chief lesions represent peripheral degenerative neuritis, and aside from rare cases of anterior cornua degeneration, the brain and spinal cord are spared, though marked cerebral symptoms are sometimes observed and a peculiar feature of lead paralysis is the *preservation of sensation* and the extraordinary frequency of *musculo-spiral paralysis*. The kidneys and gastro-intestinal tract are also frequently affected. The most characteristic symptoms are:—(a). The *"blue line"* on and within the margin of the gums unaffected by the toothbrush and representing a black sulphide of lead deposited in the papillæ. It may be faint, stippled and *blue-black* and when present is almost pathognomonic. (b). *Colic.* A violent diffuse spasmodic abdominal pain unassociated with tenderness and relieved by pressure is a common and characteristic symptom. There may be associated diarrhœa and persistent dull pain between the paroxysms. *High tension pulse* and vomiting are sometimes observed. (c). *Paralysis.* If paralysis ensues the arms are most often affected, usually bilaterally, occasionally unilaterally and the nerves of both arms and legs or of the whole body may be affected, but *the tendency is to limitation to muscle groups. Hence we distinguish a musculo-spiral variety* associated with wrist drop in which the supinator longus is usually spared. A *peroneal type* (10 to 15% of cases) involving the common extensor of the toes and that of the big toe and associated with foot drop and the steppage gait. *The brachial type* is bilateral and involves the brachialis anticus, supinator longus, biceps and deltoid, and occasionally the pectoral muscles. *An Aran-Duchenne type* is interesting because of its resemblance to the first stage of progressive muscular atrophy, the paralysis involving the small muscles of the hand and producing atrophy of the thenar and hypothenar regions. The *laryngeal form* is rare and consists of an adductor paralysis. *Various combined palsies* may be noted and general paralysis may be either of a gradual or rapid development, in rare instances simulating Landry's disease. *It should be remembered that sensation is usually preserved, a certain amount of early pain may be present, atrophy is marked, the reaction of degeneration present and the forearm tremor is commonly encountered.*

The blue line.

Lead colic.

Paralysis.

Types—musculo-spiral and peroneal.

Brachial

Aran-Duchenne.

Laryngeal.

Motor chiefly

Cerebral signs.

(d). *Cerebral Symptoms.* Hysterical manifestations, coma, convulsions, transient delirium and temporary or permanent insanity are occasionally encountered. (e). *Anæmia* is usually present in the form of a secondary anæmia of varying degree and basophilic degeneration of the red cells is constant in, though not peculiar to lead poisoning. In no other disease however do they appear in such large numbers. Wright's staining method best shows these peculiar cells (see plate II).

Anæmia and basophilia.

Renal and vascular signs.

(f). *Arterio-sclerosis* and *interstitial nephritis* are common sequences of chronic lead poisoning and the examination of the urine for lead is not only important but indispensable in many cases for the differentiation of the disease. It is especially true of those varieties which exactly simulate an anterior-poliomyelitis.

Chronic Arsenical Poisoning.—The symptoms of acute poisoning are essentially the same as those of cholera morbus with excessive abdominal pain (see page 636). Chronic poisoning possesses a complex symptomatology most baffling and misleading unless the case history suggests poisoning by arsenic. Anorexia, nausea, intermittent diarrhœa, attacks of colic or of abdominal discomfort are the usual gastro-intestinal phenomena. Chronic bronchitis may occur, as may weakness, emaciation and joint swelling. The nervous symptoms range from those of pronounced neurasthenia to neuralgia or actual multiple neuritis. The only symptom in any way characteristic is the yellowish brown pigmentation of the skin observed in advanced cases and puffiness of the eyelids. Arsenical paralysis differs from lead palsy only in its tendency to involve the legs more often than the arms.

Gastric signs and malnutrition.

Sallowness and puffy eyes.

PTOMAINE POISONING AND FOOD POISONING.—It is evident that one must distinguish between the poison inherent in the food itself, whether affecting all or only certain persons, and poisons introduced by accidental contamination in manufacturing processes, and finally, the poisons due to putrefactive alkaloids (ptomaines). Under the first class we find a large number of articles which are inherently and generally injurious, such as that caused by certain fish in tropical countries and the mussel poisoning, which however is due to a known ptomaine, *mytilotoxin*, which produces rapid pulse, dilated pupils, numbness and a cold surface. Death results from collapse in about 25% of the cases.

Poisonous fish and mussels.

ERGOTISM is a well known form due to the fungus, claviceps purpurea, which produces extensive epidemics due to the use of contaminated grain, the symptoms being either tonic, cramping, spasms, enduring hours or days and characterized by flexion of the arms, exten-

Nervous form.

sion of the legs. Marked mental symptoms amounting to actual insanity and epilepsy may terminate the convulsive attacks. A second form is characterized by gangrene of the toes, fingers or more rarely, nose and ears, preceded by muscular spasm of lesser degree, anæsthesia, paræsthesia and pain.

Gangrenous type.

PELLAGRA.—This disease has been recognized for more than two centuries in Southern Europe and occurs also in Egypt, Algiers, and Mexico. Its poison is contained in decomposed or fermented maize, whether used as a food or in the form of a stimulant of which it is the basis. Both the nature of the poison and its exact mode of development are unknown.

Symptoms.—The symptoms are general malaise, constant cerebro-spinal pain, mental irritability and insomnia which last through the winter, while the spring is associated with the full development in the form of marked dyspepsia, both gastric and intestinal, more or less severe diarrhœa or actual dysentery and erythematous rash affecting the exposed surfaces of the body and followed by desquamation. Summer brings a remission, the following spring a relapse and finally a cachexia associated with disorders of the special senses and of the mind. and spastic paralysis of the legs associated with atrophy and contracture, normal superficial reflexes and increased knee jerk. Cerebro-spinal pain is intense, associated with troublesome paræsthesia, and anæmia and emaciation are profound.

A strange disease.

Prognosis.—The disease lasts for a decade or more and furnishes a considerable proportion of the cases of insanity encountered in Italy. As might be expected the autopsy shows chronic exudative meningitis, occasionally hemorrhagic sclerosis of the posterior and postero-lateral columns and degeneration of the peripheral nerves.

LUPINOSIS.—(Lathyrism). (Chick-pea disease). This ancient disease is due to the prolonged use of the common vetch or chick-pea as food as is common during times of famine in British India, France, Italy and Algiers.

Symptoms.—With gastro-intestinal disturbance as a prodrome the onset of pain with weakness and tremor is followed by spastic paralysis of the legs with exaggerated reflexes and normal sensation. Death occurs from intercurrent disease.

MEAT POISONING.—Whether due to the ingestion of poisonous sausage, pork, beef or veal, milk or cheese, the symptoms are, in general, malaise, anorexia and nausea or certain vague pains, abdominal, cervical or general. In some instances chilliness or rigor, vertigo or

A common and serious ailment.

tremor precede the attack. The actual onset is characterized by acute gastro-intestinal disturbance resembling cholera morbus, violent abdominal pain, marked prostration, sweating, severe headache and thirst. Fever varies from 100° to 105° F., and the pulse is usually rapid.

Fig. 173. — Trichomonas vaginalis. (After Tyson.)

LACQUER POISONING.—This disease of lacquer workers strongly resembles the ivy (Rhustoxicodendrom) poisoning of this country, differing only in the greater severity of its symptoms. The base of lacquer is the balsam of the Rhus vernicifera.

Fig. 174.—Megastoma Entericum. (Grassi and Schewiakoff.)

PARASITIC DISEASES.—RHIZOPODA.—*Amœba dysenteriæ.* Is the chief member of this group and has already been discussed under amœbic dysentery, see page 281. Other forms of amœba apparently non-pathogenic are occasionally encountered in the stools, urine, mouth and lung cavities, the mode of introduction being little understood.

Fig. 175.—Cercomonas hominis (Trichomonas intestinalis). (Pribram and Davaine.)

FLAGELLATA.—The three varieties ordinarily encountered are probably non-pathogenic and merit no extended description. The pear-shaped *trichomonas vaginalis* is from 15 to 20 micra long, and 7 to 12 micra wide. Its blunt end carries a nucleus and 3 or 4 flagellæ. It is a common constituent of acid vaginal secretion. Another pear-shaped organism, *Megastoma Entericum* is 10–21 micra long, and 5–12 micra wide. From the edges of a marked depression upon its anterior surface project three pairs of cilia, another pair springing from its pointed end. The *cercomonas hominis or trichomonas intestinalis,* also pear-shaped, measures from 10–16 micra, carries 2 or 3 flagellæ, and sometimes a visible nucleus. The *Balantidium coli* may or may not be pathogenic. It is a small oval ciliated organism and carries 2 nuclei, one spherical, the other reniform. Its blunt end shows a funnel-shaped opening. It has been found on and within the intestinal mucosa and submucosa, as well as free in the stools. (50–100 micra in length.)

Fig. 176.—Balantidium coli. (After Malmsten and Leuckhart.)

TREMATODA.—(Flukes, distomiasis). The most important of these organisms is the *Bilharzia hæmatobia or Schistosomum hæma-*

tobium which causes endemic hæmaturia prevalent in Egypt, North and South Africa, Arabia, Persia and the West Coast of India. Only isolated imported cases are encountered in Europe and America. The accompanying illustration shows the peculiar relation of the sexes. The embryos are free swimming and the organism reaches the intestinal tract directly or indirectly through contaminated water. Once ingested the organism is found chiefly in the portal veins, but ultimately in other portions of the body, especially the bladder and rectum. The eggs laid in tissues may also make their way to the bladder and intestines and escape with the urine and fæces. In the tissues they may cause papillomata and fibroid processes or even form the nucleus of a vesical calculus.

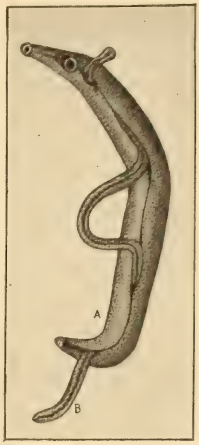


Fig. 177.—Male (a) and female (b) of Bilharzia hæmatobia. (After Looss.)

Symptoms.—The foregoing description indicates the seat, diverse nature and variety of symptoms, which are of course chiefly those of hæmaturia, abscesses and irritation of the urinary tract, dysenteric stools, rarely Glissonian cirrhosis and a varying degree of anæmia. The diagnosis depends upon the finding of the characteristic eggs.

Fig. 178.—Fasciola Hepatica. (After Wood.)

The *Faciola Hepatica* or distomum hepaticum is a small fluke measuring 20–30 mm. in length, carries fine spines and is a rare source of infection in man. Its eggs measure 130–150 micra.

The *Distomum lanceolatum* or lancet fluke is 8–10 mm. in length, and 1.5–2.5 mm. in width, and carries no spines. Infection in man is excessively rare, the intermediate host being the snail.

The *Opisthorchis Felineus* measures 8–10 mm. in length, and 1.5–2 in breadth. It is a translucent yellowish red parasite not uncommon in Siberia and occasionally found in Germany.

The *Opisthorchis Sinensis* or distomum spathulatum is like the

preceding variety in size and color, its egg measuring however 20–35 by 15–21 micra. It is abundant in India, China, Japan, and is occasionally seen in America.

The foregoing liver flukes produce a chronic disease chiefly affecting children, often members of one family and characterized by marked hepatic and gastro-intestinal symptoms, chief amongst which are irregular diarrhœa, becoming bloody, hepatic enlargement, slight fever, variable amount of pain and intermittent jaundice, anæmia, emaciation, and finally general anasarca.

The *Paragonimus Westermani* is a pulmonary fluke of a reddish brown color and oval form, measuring 8–10 by 4–6 mm., its eggs measuring 56 by 90 micra. It prevails widely in China, Formosa, Korea and Japan and is occasionally found in the United States. The symptoms produced are repeated attacks of hæmoptysis, trivial or severe, and chronic cough with rusty sputum. It should be remembered that all of the flukes found in man are flat, leaf shaped bodies with ventrally placed suckers, and that all save the Bilharzia are hermaphrodite.*

CESTODES.—(Tapeworms). These are long, flattened and segmented worms, each carrying both male and female sexual organs and lacking a digestive tract. The head or scolex carries suckers and in some varieties encircling hooks. Unless the head be removed the worm may continue its life and growth by budding. Each and every segment carries sexual organs placed along the border in the tæniæ and in the median line in the bothriocephalus. The number of eggs is enormous and they may show a developed embryo.

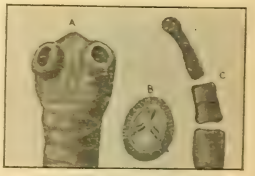


Fig. 179.—Tænia Saginata. (Mediocanellata). (After Pribram and Wood.)

Tænia Saginata or Mediocanellata is the common tapeworm of this country and Europe, sometimes called the unarmed or beef tape-worm because lacking the hooklets and frequently conveyed by beef. Its head is larger than that of the tænia solium, measuring 2 mm. or more in breadth. Its form is shown by the accompanying illustrations. Its segments measure from 17–18 mm. by 8–10. The ova are not distinguished from those of tænia solium. A length of 15 to 20 or even 30 ft. may be attained.

*The measurements throughout this section on the most part follow the descriptions given by F. C. Wood, James Tyson and Pribram.

Tænia Solium.—The tænia solium or pork tapeworm, common in Europe or Asia, but infrequent in this country. It is shorter than the tænia saginata, seldom exceeding 12 feet in length. The head is smaller, rounder and provided with double circlet or hooklets in addition to its suction discs, the neck is narrow and threadlike. The segments measure 1 cm. by 7 to 8 mm. The structure of the uterus and general form of the worm is shown in the accompanying illustration. In man and pigs the ova develop and the free armed embryos pass to different parts of the body and develop as cysticerci.

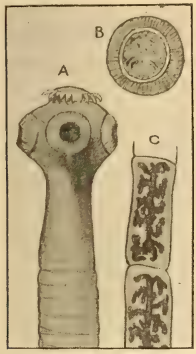


Fig. 180.—(A) Scolex, (B) egg and (C) of tænia solium. (Pribram and Wood. modified segments.)

Bothriocephalus Latus.—This cestode though largely confined to Switzerland and Japan and the coast of the Baltic is occasionally encountered in immigrants and has recently been found in Minnesota in a number of cases as reported by Wesbrook. It is conveyed by the pike or other fish; the adult worm measures 25 or more feet in length, is unarmed and carries two lateral grooves upon its head. Amongst the other forms of cestodes may be mentioned the *diplogonoporus grandis* or *krabbea grandis* found in man only in Japan, the *dipylidium caninum* common in the dog, very rare in man and also occurring in the canine and human flea and dog lice. The *hymenolepsis nana* or *tænia nana*, a small worm but 5–45 mm. in length, and 0.7 in breadth. This worm rarely found in man finds its host in the rat, its habitat chiefly in Italy. Its head carries 4 suckers and a row of from 24–28 hocklets. The egg is oval or round and measures from 30 to 37 micra, the embyro carries 6 hooks. *Hymenolepsis diminuta* or tænia flavopuncta. This small worm measuring 30–60 cm. by 3.5 mm., the head 0.2 by 0.5

Fig. 181.—Bothriocephalus Latus. (A) head (magnified), (B) egg, (C) head, neck and proglottides.

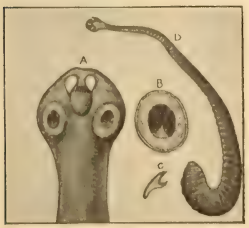


Fig. 182.—Hymenolepsis nana. (A) head (magnified), (B) egg, (C) hooklet, (D) head and proglottides. (After Brann, modified.)

mm. is armed and oval. The eggs 60–70 by 70–80 micra are yellowish and show faint radial striation. Butterflies, beetles, mice and rats are its commoner hosts.

Symptoms.—These may be those of anæmia and slight gastro-intestinal and nervous disturbances or an anæmia undistinguishable from pernicious anæmia save by finding the parasite.

Davainea Madagascariensis.—Is a rare parasite found in British Guiana, India, and Africa. It is but 30 cm. in length, and has an armed head with 90 hooks and 4 suckers. The ova measures from 6–8 micra, has two spikes and a double shell.

Tænia Echinococcus.—This interesting tapeworm inhabits the dog which becomes its conveyer to man. Australia and Iceland are the chief sources of infection, nearly half the Australian dogs being affected, and about $\frac{1}{3}$ of those in Iceland. In other countries it is rare. The adult worm consists of about 3 or 4 members and measures about 2.5 to 6 mm. in length. The breadth of the head is 0.3 mm. It carries 4 suckers and is supported by a short neck. The head is armed with a double circle of from 28 to 50 hooks. The uterus and general form are well shown on page 300.

Mode of Infection.—Direct transfer from dog to man may result from close contact especially in such countries as Iceland and Australia, where dogs are numerous and in close relation to their masters and family. Contamination of drinking water is also easily understood.

Hydatid cysts.

If the small six-hooked embryo is released through digestion of its shell it passes through the intestinal mucosa to various portions of the body, chiefly of course the liver. Wherever it lodges small double walled cysts about 1 mm. develop, the hooklets disappear and the fibrous envelope results from the slight inflammatory reaction. These primary cysts later develop secondary cysts through budding from the inner layer and these daughter cysts are structurally identical with the primary ones. Later they are released and a dozen or more may be found within the parent cyst. Each daughter cyst may itself reproduce by budding until the original cavity represents a long family line. Scolices develop carrying hooklets and sucking discs and representing the young worm and thus a multitude may arise from a single cyst. The fluid is primarily non-albuminous, limpid, and of a specific gravity from 1005–1010. The cysts may endure for many years, ordinarily undergoing a resorption and ultimate calcification, the hooks persisting even in obsolete cysts. Rupture is always possible, symptoms and danger depending upon the loca-

tion. Suppuration is a serious but unusual event, most common in the liver.

Distribution.—About 50% of echinococcus cysts occur in the liver, 20% in other abdominal organs some at less than 10% each and the pulmonary tract and nervous system.

Symptoms.—The size and location of the cysts determine the physical signs and subjective symptoms and the discovery of the hooklets in the fæces, urine, sputum, etc., makes the diagnosis positive. Obscure symptoms referable to the organs most often affected are especially suggestive in Icelanders and Australians. The hydatid thrill is almost pathognomonic but often absent even in relatively superficial cysts. It may be felt as a prolonged exquisitely fine thrill echoing the stroke of ordinary finger pleximeter percussion, but more clearly when the middle one of these pleximeter fingers is finally percussed without the usual recoil of the percussion digit. In one case observed by the author the case was symptomless, though evidently of hepatic attachment but the thrill at once solved the problem.

Hydatid thrill.

Fig. 183.—Ascaris lumbricoides. (A) female, (B) male, (C) egg, (D) head. (Pribram and Wood.)

Aside from the foregoing points a minute discussion of the host of symptoms is futile as there are none other than those of similar growths in different anatomic areas. Suppuration produces the picture of abscess, septicæmia or pyæmia.

Perforation may involve any adjacent structure and produce the symptoms peculiar to lesions of that organ; urticaria is said to be a peculiar and frequent accompaniment of perforation. Multilocular elastic or fluctuating tumors always suggest hydatids especially when in the hepatic and renal regions, but as pulmonary phthisis and gangrene, all forms of abscess, pericardial effusion, cardiac dilatation, pleuritic effusion, hydronephrosis, renal calculus, pyelitis, a distended gall bladder and even hepatic syphilis and carcinoma have been confounded with it it is evident that exploratory puncture is often required. Hooklets are seldom absent. In the central nervous system the symptoms are those of tumor. Finally it should be emphatically stated that in many cases *no* symptoms of importance appear, that in general the impairment of health and nutrition is relatively slight in the absence of complications and that recovery under operation is the rule.

Misleading symptoms.

Ascaris Lumbricoides.—This, the most common intestinal parasite requires no intermediate host and infests the upper portion of the small intestine. The male measures 15 to 17 cm., the female 20 to 25 cm., it is earthworm like in form. Being migratory they are most frequently recovered from the stools, but may be found in the appendix, the bile ducts, in vomitus or may even enter the pharynx, larynx, trachea. Eustachian tube, nasal passages or lachrymal duct. Usually but from two to ten are present, but they may exist in quantities sufficient to cause intestinal obstruction. Rare cases of intestinal perforation are reported, but are probably instances in which the presence of the worm is accidental rather than causative.

Largely gastro-intestinal or nervous.

Symptoms.—A few worms may be present without symptoms. Pallor, restlessness, irritability, nose rubbing and picking, bad breath, anorexia, colicky pains, tympanites, malaise, impaired nutrition, disturbed sleep with teeth grinding. Complex and misleading nervous symptoms such as epileptiform seizures, general convulsions, tetany, vertigo, aphasia and even actual paralysis, localized or hæmiplegic, as well as recurrent febrile attacks are among the symptoms attributed to this parasite. As a matter of fact each and all are merely suggestive and the diagnosis can never be made unless the worms or their eggs are found. They are excessively rare in infancy and most common from the 3rd to the 10th year.

Not distinctive.

Oxyuris Vermicularis.—The "pin" or "thread" worm measures 4 mm. for the male, 10 mm. for the female. These migratory parasites infest the folds of the lowest bowel, seldom being found above the cæcum, though rarely they migrate as far as the pharynx or perforate the intestine and form "verminous tubercles." In the stools they resemble bits of white thread and are readily dislodged by appropriate medication. The prominent symptoms are genital and anal itching with nocturnal exacerbations and such other symptoms as have been described as associated with the round worm, save the nervous symptoms are less prominent and mucous colitis and such reflex irritation symptoms, frequent urination or incontinence and balanitis, vaginitis and perhaps masturbation are common. Here also the worm or its ova are essential to diagnosis, and are easily found, often indeed about the *external* genitals or anus. The worm is conveyed from person to person or by infected water or food.

Fig. 184.—Oxyuris vermicularis. (A) female, (B) male.

Itching.

Nervous symptoms.

TRICHINIASIS.—In man trichiniasis depends upon the ingestion of infected, raw or imperfectly cooked pork. Those suffer most who eat raw and underdone meat, and most of the cases encountered in this country have been Germans. It cannot be a rare condition as Williams reports 5% as showing infection in a series of 500 autopsies, but it is often unrecognized. The larvæ require about 72 hours of residence in the intestines to become sexually mature and at the end of a week each female may have discharged into the lymph spaces or blood stream several hundred embryos. In due course these pass to the muscle fibres in which they create a reactive inflammation and become encapsulated. They may or may not so live for years. In man the appearance of an old cyst is that of "an opaque oat shaped body" (Osler) owing to late calcification.

Underdone or raw pork.

Prolific females.

Symptoms.—Assuming that a sufficient number of Trichinæ have been ingested symptoms appear in about one week and are usually characterized by the conjunction of acute gastro-intestinal disturbance, fever, usually remittent or intermittent, followed by muscular pain and tenderness, limitation of movement, edema of the face, extremities and surface over affected areas and a marked leucocytosis in which the eosinophiles often reach 50 to 70% of the total leucocytes. The attention should be fixed upon the muscle symptoms and eosinophilia, as the case may assume the typhoidal form, be complicated by albuminuria, pleurisy, pneumonia and dysphagia, aphonia, etc., etc. In other instances symptoms are of the scantest. The mortality is extremely variable (2 to 30%). The diet and the grouping of cases may also be suggestive, but oftentimes the muscles must be directly examined if the stools, ingested food and other more available material fail to show the organism. Harpooning the muscle is neither necessary nor proper if local anæsthesia and a clean incision can be employed. Many cases are mistaken for rheumatism in spite of the lack of joint involvement and the peculiar muscle symptoms.

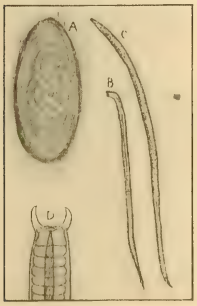


Fig. 185.—Trichina spiralis. (A) encysted in muscle, (B) male adult, (C) female adult, (personal observation) (D) male genital apparatus.

Muscle pain and tenderness.

Puffy face.

Eosinophilia.

Mortality.

Direct diagnosis.

UNCINARIASIS.—(Ankylostomiasis, Egyptian chlorosis). Once regarded almost exclusively as a tropical disease uncinariasis has been proven widespread and by no means limited to the tropics, though more

widely prevalent in southern latitudes. It may be found in Egypt, England, Europe, Switzerland, India, the Malayan Archipelago, the West Indies, and our Southern states furnish frequent examples. Stiles has described an uncinaria Americana differing slightly from the older form (ankylostomata duodenalis). The dimensions of both are for the male 7–11 mm., for the female 10–18 mm. (see plates). No intermediate host is necessary and the organism thrives in dirt and puddles. There are many reasons for believing that the skin may be a channel of infection. Adult worms may be found in any portion of the gastro-intestinal tract, but chiefly in the duodenum and rarely in the stomach or colon.

Fig. 186.—Uncinaria (ankylostoma) duodenale. (A) female, (B) male, (C) eggs, (D) male and female of natural size. (Pribram, slightly modified.)

Symptoms.—There may be none in cases showing the worm in the fæces, but ordinarily gastro-intestinal symptoms co-exist with anæmia, simple and mild, or so profound as to imperfectly simulate pernicious anæmia from which it is distinguished by normoblast predominance and a low color index. Stiles emphasizes the peculiar lack-lustre stare and the muddy or waxy white skin. The remaining symptoms are those of profound anæmia with anæmic bruits, breathlessness, late edema and a peculiar ascites. The characteristic feature of the anæmia is the excess of eosinophile cells both relative and absolute, most marked in early cases. The proportion of these cells to the whole leucocyte count varies from 4 to 50%, the average being between 20 and 25%. A rise in the count under treatment is a good omen, a low original count or a falling one is a bad one. Leucocytosis is present in a majority of the cases, but is seldom high, a count above 12,000 or 13,000 being exceptional. The more advanced the disease the lower is the leucocyte count. The color index is as a rule low and in the tropical cases especially so, the same being true of the red cell count. Cell deformity is marked in the severer cases but normoblasts predominate.

Eosinophilia. Color index low.

FILARIASIS.—This extraordinary parasite includes three chief species. The *Filaria Bancrofti*, also called Nocturna, is found in the peripheral blood only at night or during sleep, whereas the *Filaria diurna* is found only during the waking hours or in daylight. The *Filaria perstans* is present day and night and is said to be present in from

Three chief forms.

50–90% of West African negroes, being apparently non-pathogenic The Filaria demarquaii is found in the West Indies, persists day and night and is actively motile. The Filaria Ozzardi found in British Guiana and a large species, the Filaria Gigas, has been found at Sierra Leone. The first two forms, viz.:—nocturna and diurna are the only ones of clinical importance. Filaria nocturna is found in all tropical and many sub-tropical countries. Most of those observed in the northern part of this country are imported cases. In certain tropical regions 70% of the population of certain villages may be affected.

Sub-varieties.

Morphology.—*Filaria Nocturna.* The parent worm inhabits the lymph channels, the embryo the circulating blood, the body of the mosquito serving as intermediate host. The adult form is from 3 to 4 inches in length and of the thickness of a fine thread. The embryo is 1-80 of an inch in length and about 1-3,000 of an inch broad, delicate and transparent and enclosed in a transparent sheath. If the blood of a sleeping patient be examined day or night, or that of a waking patient by night, the microscopic findings are usually simple and definite. If a large drop, a broad smear, or, a large wet preparation be taken, no staining is necessary and low power lenses are sufficient. Both culex and anopheles may act as intermediate hosts.

Fig. 187.—Filaria Nocturna embryo of filaria Bancrofti.

Peculiar habits of parasite.

Symptoms.—No symptoms appear in the majority of cases, the embryos indeed being innocuous. The parent worm produces at times either lymphatic varix or edema from lymphatic obstruction, hence there are produced a group of symptomatic conditions of lymphatic origin. (a). *Hæmatochyluria.* Chylous urine always suggests filariasis. Its appearance is described on page 314 and its cause is a leakage of chyle usually from the lymphatics around the kidney pelvis or bladder. The urine coagulates on standing and the contracted white clot ultimately floats in the milky liquid. The condition is almost symptomless, though rarely clots may cause retention, or loin weariness may exist. (b). *Lymph scrotum* is merely lymphatic varix of the scrotal channels and painless soft tumors, usually in both groins also occur through varicose lymph glands. In either condition pain indicates lymphangitis. (c). *Elephantiasis and Elephantoid Fever* These conditions occurring in

Often absent.

Chyluria.

the tropics are no doubt generally due to the Filaria, but this is not true of the elephantiasis of the temperate zone. The symptoms are chill, high fever, heat, redness and congestion over the area of lymphatic involvement with inflammatory induration which persists after the crisis. Abscess sometimes occurs. The specific pathogenic activity of the Filaria diurna is not yet established.

DRACONTIASIS.—(Guinea worm disease). *The Dracunculus or Filaria Medinesis.* The male worm is unknown, the female, cylindrical and smooth, measures 50 to 100 cm. by 2 to 3 mm. and carries a blunt hook on the head. The embryos are 500–750 micra by 25–30 and live in moist earth or muddy pools, finding an intermediate host in a small aquatic Cyclops. It is found in India, Persia, Arabia, Africa and Brazil. Rare cases have been reported in this country. Its preferred site is the subcutaneous tissue of the foot and leg, where it forms ulcers from which the head may protrude. Nevertheless, the stomach is the portal of entrance, the worm penetrating the intestine and traveling downward to the lower extremity. If hot water be poured upon the ulcer the embryos may in part be discharged and after spontaneous parturition the worm may voluntarily come forth.

Trichocephalus Dispar.—(Whip worm). This denizen of the cæcum and colon measures from 40 to 50 mm. and has a thread-like anterior, and thicker hinder portion, conical and pointed in the female, obtuse and often coiled in the male. The eggs, 0.05 mm. in length, carry a bud-like projection. The only pathogenic symptom associated with it is anæmia with enteritis, and this is rare.

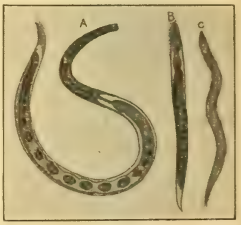


Fig. 188. — Strongyloides intestinalis. (A) female, (B) rhabditiform larva, (C) filiform larva. (After Braun.)

Dicotophyme Gigas or Eustrongylus Gigas.—This foot long worm, usually seen only in animals, is occasionally found in the renal region of man.

Strongyloides Intestinalis.—This small worm measures 2 mm. by 0.06 mm. It is frequent in Southern China and has been found in Manila, Germany, Italy and America. If present in great numbers they cause enteritis and anæmia.

Psorospermiasis.—Psorospermes gregarenidæ or psorazoa are occasionally found in man in connection with obscure febrile ailments. In the cases reported the lesions resemble the granulomata but are found to contain coccidia.

TRYPANOSOME FEVER AND THE SLEEPING SICKNESS.

—The former is an irregular fever characterized by swelling of the spleen and lymph glands, weakness and emaciation. The latter is a peculiarly latent and fatal disease native to West Africa, characterized by early apathy, mumbling, hesitant speech, tremor and difficult locomotion. The latent period may last five or more years, the increasing drowsiness becomes a deep and continuous sleep or coma and the patient dies ordinarily of septic meningitis. The disease is fatal, occasionally attacks Europeans and usually lasts from three months to a year, dating from the commencement of active symptoms. The chain of proof is not complete, but it is certain that these organisms are uniformly present in this disease.

Fever.

Sleeping sickness.

Course.

Fig. 189.—Trypanosoma hominis. (Dutton and Laveran.)

The form of the organism is shown in the accompanying plate. It is small, actively motile, non-pigmented and transparent, measures 10–20 micra and carries a curious flagellum on one side of its body. It is extra-corpuscular and possesses an oval nucleus and a centrosome nearly opposite the attachment of the flagellum. It apparently multiplies by fission. The carrier of infection is a species of Tsetse fly.

Parasite.

Tsetse fly.

PYROPLASMOSIS HOMINIS.—Wilson and Chowning believe the Pyroplasma hominis to be the cause of the curious and fatal disease prevalent only in small districts of western Montana and particularly in a small area from 4–10 miles wide and 50 miles long in the western side of the Bitter-root river. It prevails only from the middle of March to the middle of July and seems to affect only those persons who have been bitten by ticks. Dr. Chowning states that these were found in every case but might easily be overlooked because at times only present in the hair about the genitals. The organism is both extra and intracorpuscular, the bodies are small, hyaline, resemble the malarial fever hyaline forms, but at no stage carry pigment. No ameboid movement has been observed during the first stage of its development, but it is actively ameboid during the second stage. It is readily found in the capillary blood of congested areas and the maximum corpuscular infection occurs in the spleen, liver, lungs and kidneys. The later investigations of Chowning

Limited range.

Ticks.

Resembles hæmameba malariæ.

Not pigmented.

and Anderson seem to clearly substantiate the claims first made, which were seriously questioned by Stiles and others.

Symptoms.—After an incubation period of from 3–10 days, headache, nausea, muscular soreness and a chill or chilliness and nose bleed are followed by a rapidly rising fever and unduly rapid pulse and respiration. The rash appears on the third day, first on the forehead and extremities, later on the chest, abdomen and back. It is macular, bright red and becomes petechial in severe cases. It is profuse everywhere but on the abdomen and desquamation may follow. In fatal cases rapid anæmia appears with sustained high temperature and a rapid weak pulse. Albuminuria is constant.

Peculiar rash.

Albuminuria.

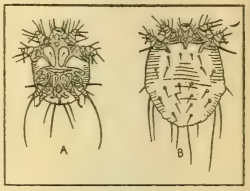


Fig. 190.—Sarcoptes scabiei. (A) male, (B) female. (Braun.)

Differential Diagnosis.—No confusion can arise if the salient points are recalled. The history of exposure, of tick bites, of discovery of the tick itself, its sporadic nature, the distribution, character and time of appearance of the rash are sufficiently distinctive.

Prognosis.—In favorable cases termination by lysis is initiated by the 12th day. The mortality varies from 70–90% and pernicious cases of rapidly fatal course are seen.

Sarcoptes Scabiei or Acarus Scabiei.—The female is the active agent, the male usually absent. The habitat is a burrow in the epidermis, usually of the axilla, front of the abdomen and more commonly the finger and toe webs and the lateral opposed surfaces of the digits. Characteristic scratch marks and erosions direct attention to the burrows, which appear as short dark lines leading to a shining spot. The female, which may be seen with the naked eye, may be removed by passing a needle along the black line of egg deposit to the spot which indicates her presence.

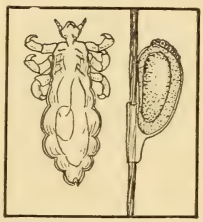


Fig. 191.—Pediculus capitis and egg.

Pediculus Capitis.—Produces irritation chiefly about the posterior margin of the scalp and the eggs or nits indicate by their position in the hair the duration of the process.

Pediculus Corporis.—Should be sought in the seams of the clothing, and the bites are dark, centered, hemorrhagic spots with a pale areola. Scratch marks are of course common, chiefly over the upper back and shoulders. Every hospital interne in a public service gets to know them.

Pediculus Pubis.—This should be sought in and about the hair of the genital region, but is occasionally found in the axillæ and the eyebrows.

The *maculæ ceruleæ* (tache bleuâtre) are subcutaneous bluish spots from 5–10 mm. in diameter, upon the abdomen and thigh, and are due to the irritation of body lice.

Vagabond's disease is a deep pigmentation resembling that of Addison's disease, but due to the constant irritation and scratching produced by the continuous presence of body lice.

Fig. 192.—Pediculus pubis.

Cimex lectularius (bed bug) and **pulex irritans** (flea) need no extended description.

The sand flea or jigger (Pulex penetrans) is especially frequent in the West Indies and South America. It usually produces pustules or vesicles in the skin of the feet under which it grows.

ACUTE RHEUMATISM.—This is the most important of the group of acute infections of the joints and possesses a sufficiently definite syndrome and marked tendency to specific complications to justify its consideration as a separate disease, though up to the present time no one of the numerous organisms associated with it has been proven causative.

Germ unknown.

Etiology.—The disease is especially common in cold, damp, changeable climates, is most common in spring and fall, frequently associated with occupations involving exposure to wet and cold, particularly if combined with impure air, exhaustion, improper dietary or bad habits. It is said to have followed nervous shock and severe physical injury and to be especially common in neurasthenia and anæmia. Its frequent association with chorea is notable and it is the most common source of and hence frequently associated with acute and chronic valvular disease of the heart. At times it seems to be epidemic though there are usually related peculiarities of the atmospheric sort. Its strong resemblance to sepsis is noticeable.

Climate and season.

Relation to shock and injury.

Symptoms.—The *onset is usually abrupt* and is preceded by *tonsillitis* in about ⅓ of the cases. Chilliness or perhaps a rigor is succeeded by a rapid rise in temperature, associated with painful and swollen joints, perhaps showing the outline of the distended synovial membrane, the involvement being usually bilateral or symmetrical in its advance, and the larger joints, especially the ankle and knee, being usually first affected. Occasionally there is

Onset.

Symmetrical.

Larger joints chiefly.

universal joint involvement constituting one of the most atrociously painful of all diseases. The patient is usually rigidly quiet and apprehensive, suffering from the lightest touch or jar. The migratory tendency of the disease is marked; it is accompanied by acid sweats of a peculiar and characteristic odor and anæmia is invariable and rapidly produced. Hyperpyrexia is an occasional and unfavorable symptom and endocarditis, pericarditis and less often myocarditis are the commoner complications. In these cases the heart should be examined carefully at every visit and any irregularity in the pulse, precordial pain or oppression, or sudden accession of temperature without the involvement of new joints, should lead to an especially careful examination. Pneumonia and pleurisy must also be considered as occasional complications. The disease usually terminates by lysis and under modern treatment seldom lasts for more than two or three weeks though with the older treatment the saying that a cure for rheumatism was "*six weeks*" accurately expressed both the inadequacy of the earlier methods and the average self limitation of the disease. It is peculiarly recurrent in tendency, especially if the anæmia and general malnutrition accompanying it do not receive attention.

Sweats and odor.

Anæmia.

Cardiac lesions.

How indicated.

Termination.

Tendency to recurrence.

Differential Diagnosis.—This is seldom difficult if one remembers the tendency to the involvement of the larger joints, the usual bilateral and symmetrical invasion of joint surfaces, the accompanying acid sweats and the absence of septic foci. As will be seen *gout* tends to involve the smaller joints, the ordinary *septic arthritis* has usually a definite focus, is secondary to an acute infectious fever, is more frequently monarticular and shows a tendency to suppuration. *Tuberculosis* is usually a chronic process of a well defined type and the one disease which cannot be differentiated positively in all cases is the *acute atrophic arthritis* which may be suspected if in any case an acute multiple arthritis of the rheumatic type primarily involves the smaller joints or proves unduly persistent and resists the well known drugs which are practically specific in the treatment of rheumatism.

Gout, septic arthritis and tuberculosis.

Acute atrophic arthritis.

Other Forms of Infectious Arthritis.—In scarlatina, influenza, syphilis and gonorrhœa arthritis may occur, all varieties being distinguished by the rarity of osseous involvement or subsequent anchylosis. Aside from rheumatism so called, the tendency is to monarticular involvement or in the purely septic polyarticular form, to the rapid appearance of suppuration, this being especially true of the relatively common gonorrhœal form which is of a chronic and persistent type. Those acute forms associated with the exanthemata and acute infectious

Monarticular.

Suppurative.

diseases are especially common during convalescence and may or may not suppurate, their true nature being suggested by their association. In typhoid there is a special tendency to a similar involvement of the spine.

Typhoid spine.

Tuberculous arthritis cannot be fully considered. It is marked by a slowly progressive, low grade inflammation with the lack of outward signs of inflammation giving it in the knee joint the name of "white swelling," though heat, pain and tenderness may be present. The X-Ray shows involvement of the bone, there is a tendency to the formation of sinuses from pus accumulation, muscular atrophy is marked and spasm and deformities common.

White swelling.

In **hip joint disease** there is limitation of movement, shortening, muscular spasm and a tendency to the formation of abscesses opening in the upper thigh or posteriorly in the region of the great trochanter or below the gluteus maximus.

SUBACUTE RHEUMATISM.—All grades of rheumatic involvement may be encountered, with or without a history of succeeding acute attacks. All the symptoms are mild and frequently the chief manifestations are in the sheaths of the tendons. Such cases are peculiarly obstinate, septic in their manifestation, yet lack suppuration, are especially prone to recur and often unusually resistant to anti-rheumatic remedies.

Resists treatment.

GOUT.—(Arthrosia podagra). **Etiology.**—One of the older writers has said that gout is the daughter of limb relaxing Bacchus and Venus, another has pronounced it a sort of patent of nobility or at least a proof of gentle descent but this is far from being true as numerous cases of "poor man's gout" attest. Of the actual pathology we know next to nothing, it having once constituted a famous battle ground but never with decisive results. We know that a sedentary occupation, high living, the male sex, the middle age period, hereditary influence (with many alternatives in inheritance), lead poisoning, depressing emotions, overwork, traumatism, passion and other similar conditions are directly or indirectly related to its appearance in the individual.

"Poor man's gout."

Symptoms of Acute Gout.—The onset is sudden, usually at or after midnight and the great toe is the usual seat of the intense pain, followed by a red or purple, tense, shiny, edematous and exquisitely tender swelling. Other joints may be involved primarily or secondarily the preference being almost invariably for the smaller. The urine is scanty and high colored, sharply acid, and contains an excess fo urates. The attack subsides during the latter part of the day

Sudden onset.

Intense pain.

Rapid swelling.

Course.

only to come on again the succeeding night or at irregular intervals with even greater intensity, lasting oftentimes for 8–14 days unless the treatment is radical and effective. In the so called retrocedent gout the storm centre may be transferred from the toe to the stomach, heart, intestines, brain, or even as in a case known to the author, to the nose.

Irregular Gout.—A large volume would be required to discuss the manifestations of this *bête noir* of the physician and victim. In Great Britain it is as shifting and misleading a condition as is malaria in our Southern states and as convenient a term to cloak uncertainty as was the old "blasted and planet" of the early times. It is certainly true, however, that gout may affect any portion of the body in its irregular form and thus we find gastro-intestinal, cardiac, renal, nervous, respiratory and cutaneous conditions unquestionably related to gout as a primary cause. Amongst the ailments thus classed may be named recurrent abdominal colics, moderate or severe dyspeptic attacks, gravel, irritable bladder, gouty kidneys, non-specific urethritis, intractable eczema, urticaria, herpes and pruritus, pharyngitis, laryngitis, bronchitis, asthma, iritis and conjunctivitis, arterio-sclerosis, phlebitis, tachycardia, a form of diabetes, and more or less profound neurasthenia and melancholia as well as persistent and severe neuritis, migraine and neuralgia. Such conditions may occur independently of acute attacks or apparently substitute them and unfortunately are almost as likely to appear in the steady-going descendant as in the high living ancestor.

A convenient term.

Yet a real disease.

Multiform nature.

ARTHRITIS DEFORMANS.—This chronic and obscure disease of the joints chiefly affects women, and in them is frequently associated with the menopause. Heredity seems to play a part, but not in connection with a specific joint disease such as gout or rheumatism. The disease is distinctly suggestive of a nervous origin, though chronic infection cannot as yet be excluded. It is most common between the ages of 30 and 50, but not rare in younger persons and differs from gout in the absence of arthritic deposits of sodium urate and from chronic rheumatism in its marked atrophic changes in the cartilages and bones or hypertrophy or over-growth. The three recognized types depend upon both pathologic and symptomatic differences and we recognize a predominance of changes in (a) *the periarticular and synovial structures*, (b) *predominating atrophy of bone and cartilage*, (c) *hypertrophy*. Osseous proliferations occurring at the joint margins are known as osteophytes, or on the knuckles Haygarth's nodosities. Any joint and even the whole spine may become anchylosed. Marked deformity

Affects women chiefly.

Differs from gout.

Three types.

Haygarth's nodosities.

and muscular atrophy and contractures are often associated with neuritis and trophic changes. The lateral nodules frequently seen on the distal phalanges are known as Heberden's nodes. They are seldom or never associated with cases involving the larger joints. Single joints may be involved, the spinal column, shoulders, hip and knee being the common sites. This form is most common in men and at advanced ages. The spine may be involved alone (spondylitis deformans, "poker back") associated with ascending degeneration of the cord, and pain, muscular atrophy and anæsthesia due to involvement of the nerve roots. If the hip and shoulder joints are also involved (spondylose rhizomélique) the nervous symptoms are moderate or absent and the whole or only a part of the spine may be involved in certain instances.

Heberden's nodes.

Spondylitis deformans.

Spondylose rhizomélique.

General Progressive Arthritis Deformans.—The acute form exactly simulates subacute rheumatism and differentiation is at first impossible. The absence of a complicating endocarditis and the persistence of the inflammation in the joints first affected, together with the lack of reaction or response to anti-rheumatic medication are points of some value, strengthened by the later development of crepitation. Mental depression and emaciation are marked and the attacks frequently bear a relation to lactation and child-bearing. The chronic form usually succeeds one or more acute attacks and there is pain on movement, inflammatory swelling and a variable amount of effusion. The tendency is to gradual progression and symmetrical involvement, the hands, knees and feet being usually the earliest points of attack. Pain may be moderate or extreme and of the neuritic type. Crepitation appears, there is marked deformity as shown in figure 4, and finally a firm anchylosis due to periarticular infiltration and adhesion. The muscular atrophy, usually that of disuse, is nevertheless marked and may be so rapid as to suggest a central cause, which presumption is strengthened by the glossy or pigmented skin, onychia and paræsthesia. The anæmia may be marked and the disease may render the patient absolutely helpless.

Acute form like rheumatism.

Exhaustion and malnutrition.

Pain variable.

Crepitation and anchylosis.

Trophic symptoms

The ordinary form of chronic rheumatism associated with a history of previous rheumatic attacks and lacking as a rule any extreme deformity or anchylosis is distinctly affected by weather changes and usually of relatively slight importance. In gouty arthritis there are deposits of sodium urate in the soft parts which lie just beneath the skin and often perforate it, differing from the nodules of hypertrophic arthritis in their mobility. The apparent deformity is often very marked, the actual destruction relatively slight.

Chronic rheumatism.

Chronic gout.

THE NERVOUS SYSTEM AND ITS DISEASES.

Neurone theory.

STRUCTURE.—*Though incomplete, the prevailing theory concerning the structure and functional activity of the nervous system has the merits of simplicity and ready adaptation to known symptomatology.* As now conceived the nervous system is an aggregation of like units, each consisting of an *excitable ganglion cell* and cellulifugally (from the cell) conducting *axis cylinder* process of variable length (*axone, neurit*), which running naked or becoming ensheathed *as a medullated nerve fiber*, gives off lateral branches (collaterals) which, like its terminal, split ultimately into fine fibrillæ known as *end brushes* or *arborizations*. The cell also has cellulipetally (to the cell) conducting protoplasmic processes known as *dendrites*. With relation to the cell *these represent respectively the transmission and reception of outgoing and incoming impulses*. Although the terminal axone arborizations of one neurone are intimately related to the dendrites of another it is probable that there is no actual connection, but rather mere contact or contiguity. The function of the basic plasma is unknown but in the cell body are compact coils of fibrillæ which are regarded as the true conducting elements and certain basophilic granule groups known as Nissl's tigroid substance, which are so modified or diminished under conditions of cell fatigue or disease as to suggest a trophic function.

Ganglion cells.

Axis cylinder.

End brushes.

Dendrites.

Fibrillæ.

Chromophilic granules.

Mode of Action.—If one assume an afferent (sensory) impulse as originating in any of the peripheral special sense organs (sensory dendrites) and considers the nature of the conducting mechanism he can readily appreciate the facility of transmission from one set of neurones to another until a reflex or voluntary centre is reached and the equal readiness with which its efferent (motor) fibre carries the message. This marvelous telegraphy is the basis of all acts, reflex, voluntary or automatic, and involves a maze of complicated pathways and substations which relate to the orderly association and interaction of sensation, movement and intellection necessary to human life and normal activity. *Regional distribution and function are both conserved by the grouping of cells and fibres to form sensory and motor tracts, reflex pathways, trophic cells* and *primary governing centres.*

Conduction and transmission.

Regional grouping.

Conduction in Motor and Sensory Areas.—*Motor impulses* originating in the pyramidal cells of the cortex pass through their axones to a terminal arborization in the *anterior horn of the spinal cord or to a cranial nerve nucleus* where they are received by the dendrites of the related neurones and through their axones conveyed to the periphery. *The sensory impulse* requires in addition to these central and peripheral

Motor impulses.

Sensory impulses.

neurones certain "*parenthetic*" *neurones* hence the impulse from the skin passes through the *dendrites* of the peripheral neurone to the spinal ganglion and through its axone to the cells of the cord or to the medulla oblongata, and from these cells, acting as *parenthetic neurones*, to the

Parenthetic neurones.

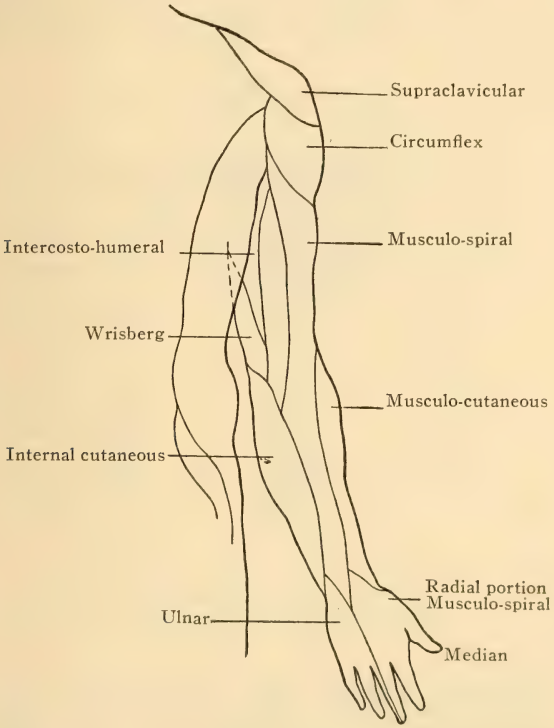


Fig. 193.—Distribution of the sensory nerves of the skin of the arm, posterior aspect. (G. R. Butler.)

cortical or central sensory neurones. *Stimuli conveyed from the centre to the periphery are called centrifugal, those carried from periphery to centre centripetal*, hence in general, "centrifugal" corresponds to motor and centripetal to sensory impulses.

Centrifugal vs. Centripetal impulses.

Reflexes.—*A reflex consists in the transmission of an impulse along a sensory neurone to the reflex centre, and thence along the motor axone*

Defined.

*to the muscle.** It requires, therefore, an unbroken chain from the periphery to the centre and back to the periphery, and excessive activity is prevented by a cortical governing (inhibition centre). A reflex may be *localized and co-ordinate*, in that the primary stimulus produces movement in a single muscle or in a limited group of muscles, or *inco-ordinate or spasmodic*, and either general or confined to certain muscle groups. Such are seen in general convulsions from various causes or

Inhibition.

Co-ordinate vs. Inco-ordinate.

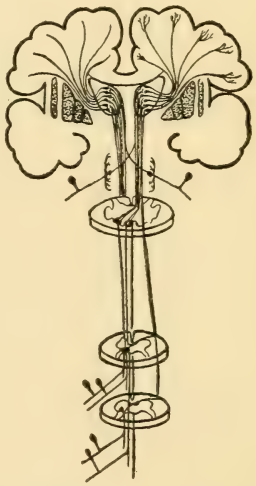


Fig. 194.—Direct sensory tract. Course of fibres and general arrangement of neurones. (After Van Gehuchten.)

a reflex spasm associated with certain diseases, and yet another form of reflex may be both co-ordinate and purposeful. Reflex centres exist in the spinal cord, pons varolii and medulla, the former being largely related to muscular action, the latter to the vital but unconscious and automatic vital functions, i.e. cardiac, respiratory, etc.

Automatic.

Cortex.

The Motor Tracts.—The *direct motor tract* arises in the *cortical cells constituting the motor areas* (see fig. 203), and *its neuraxones pass through the corona radiata* and are gathered into a narrow band occupying most of the *posterior segment of the internal capsule*; thence they pass

Corona radiata.

*It is primarily a centripetal sensory impulse conveyed to the reflex centre and there converted into a centrifugal or motor stimulus.

through the crura cerebri to the *pons varolii and medulla, giving off in the latter collaterals which, with the exception of the sixth nerve, cross the median line to the nuclei of the motor cranial nerves of the opposite* Internal capsule, pons and medulla.

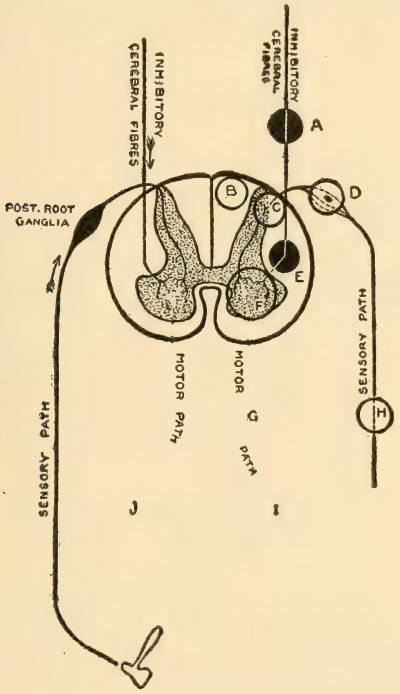


Fig. 195.—Diagrammatic representation of the reflex arc. Normal on left, interrupted on right. It will be noted that the arc proper extends from the percussion hammer (sensory stimulus) through the peripheral sensory nerve, through the ganglion of posterior root, through the posterior to the anterior horn where the sensory impulse is translated into a motor response which in turn is modified by the inhibitory cortical fibres. It is evident that a lesion interrupting the arc at either C, D, G, H, or F, will abolish the reflex, and produce flaccid paralysis if the nerves themselves or the cells of the anterior horn are seriously involved. Spastic paralysis, if the lesion be at E or A with consequent exaggeration of reflexes through loss of cerebral control (lateral sclerosis cortical lesions). Lesions at C and D cause loss of knee jerk and associated with a lesion at B represent the usual seat of locomotor *ataxia*. (After Gowers, Butler, Herter, et al.)

side; the main bundle in passing through the medulla sends nine-tenths of its fibres across the median line at its lower portion to form the *motor decussation and the anterior pyramids*, thus the original bun- Decussation.

Crossed tract.

dle is divided into *uncrossed* and *crossed* columns. *The crossed fibres enter the lateral column* of the spinal cord to form the *crossed pyramidal tract* which descending, diminishes in size as it yields fibres at different levels (spinal segments) to form the spinal motor nerves, in each case *ending in a terminal arborization about an anterior*

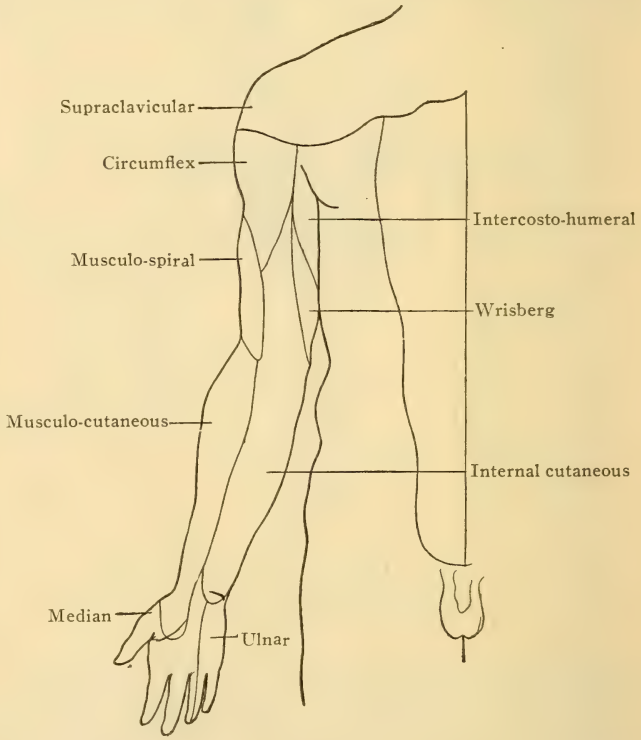


Fig. 196.—Distribution of the sensory nerves of the skin of the arm, anterior aspect. (G. R. Butler.)

horn cell which transmits the impulse through its own neuraxones (spinal nerves). *The uncrossed tract forms the column of Türck* (direct pyramidal tract) and *its fibres cross* in the *anterior commissure* at the different levels, to participate in the formation of the spinal motor nerves through an arborization around the anterior horn cells, exactly as in the case of the crossed pyramidal tract fibres.

Direct tract.

Motor nerves.

The *indirect motor tracts* follow primarily much the same course but after giving off terminal arborizations to the pons-nuclei cross the median line in the middle cerebellar peduncle, thence they pass to the cerebellar cortex, and thence after arborization to the lateral fundamental column and anterior horns. They are related to co-ordination and higher reflex and automatic movements.

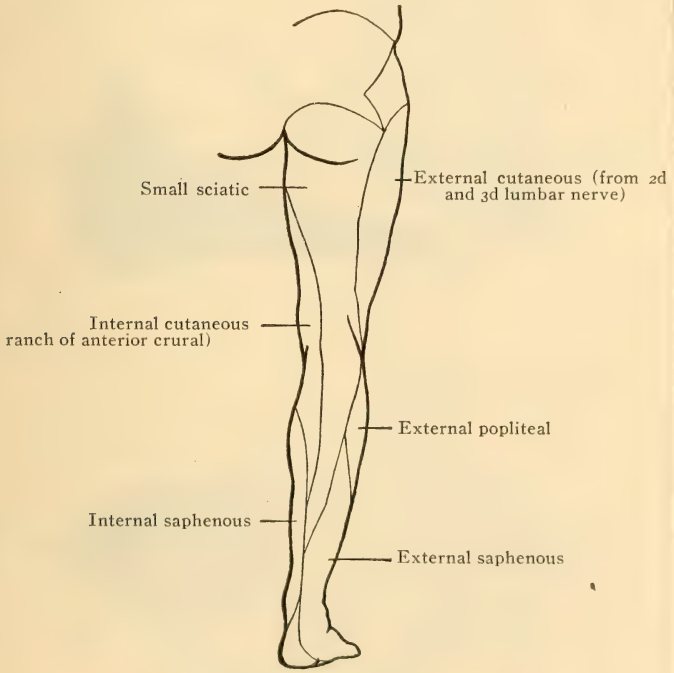


Fig. 197.—Distribution of the sensory nerves of the skin of the leg, posterior aspect. (G. R. Butler.)

Direct Sensory Tract.—From the *skin* the impulse passes along the *peripheral nerve* and to its cell in the posterior spinal ganglion, thence to cells in the posterior horns which receive the stimulus and for the most part transmit it by their neuraxones across the cord through the anterior commissure to the opposite antero-lateral ascending tract, and through the medulla and pons to the optic thalamus where another cell receives and by its neuraxone transmits it to the cortex.

Pain, touch, temperature sense.

Visceral sensation and co-ordination.

The indirect sensory tract *conveys stimuli from muscles, joints and the viscera through the posterior root ganglia and thence along the sensory roots to the cord* where a part *enters directly*, and ascends *in the posterior* column of the cord of the same side as far as the *nuclei of the columns of Burdach and Goll* (nucleus cuneatus and nucleus gracilis) in the medulla *from which* centres the *neuraxones* (internal arcuate fibres) cross in the *sensory decussation.* Some go thence to the cerebellar

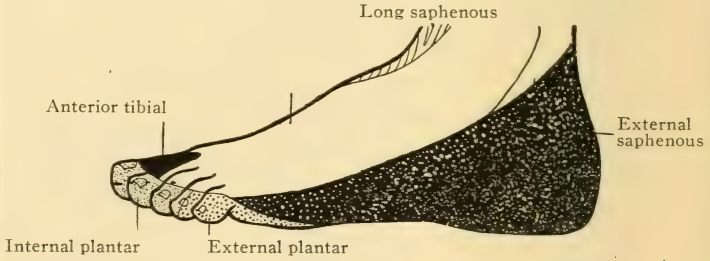


Fig. 198.*

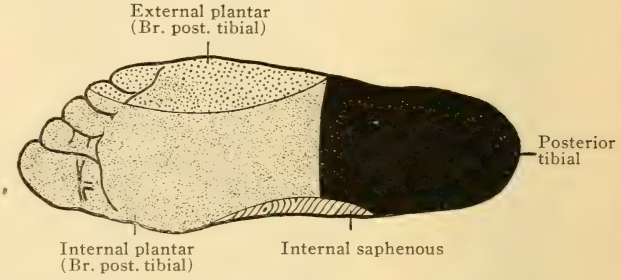


Fig. 199.*

*Figs. 198, 199.—Distribution of sensory nerves of the skin of the foot. (G. R. Butler.)

cortex, whence they are transmitted by cells of that area through the superior cerebellar peduncles to the optic thalamus and red nuclei, and thence by yet another neuraxone to the central convolutions. Other impulses pass from the sensory root to the cells of Clark's column and pass up the direct cerebellar tract to the cerebellum, thence to the optic thalamus, red nuclei and cerebral cortex.

Functions of the Sensory Tract.—*The direct* sensory tracts represent chiefly *pain, touch* and *temperature sense, the indirect* sensory and motor tracts are intimately connected with co-ordinate movement, voluntary or involuntary and, of course with visceral sensation. These wonderful tracts govern the automatic and psycho-reflex acts of the human organism.

Fig. 200.*

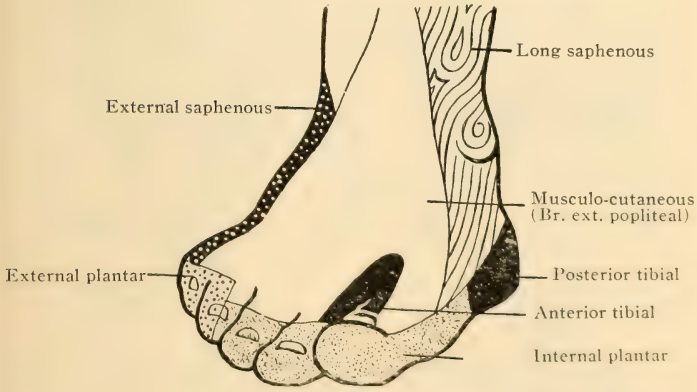


Fig. 201.*

* Figs. 198, 199, 200, 201.—Distribution of the sensory nerves of the foot. (G. R. Butler.)

Functions of the Tracts of the Spinal Cord.—The functions of the various tracts of the cord are, so far as known as follows:—The *direct pyramidal tract* is purely motor and its fibres cross at the level of the nerve emergence. The *crossed pyramidal tract* is the chief motor

Motor tracts.

area of the cord representing the fibres which cross in the lower portion of the medulla. The peripheral nerves of both tracts are dependent upon

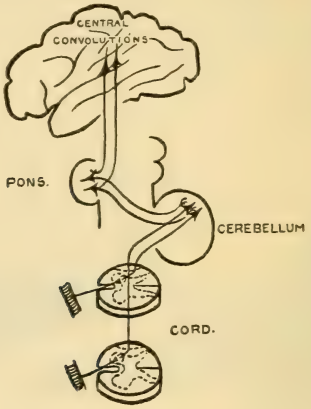


Fig. 202.—The indirect (involuntary) motor tract. (After Van Gehuchten.)

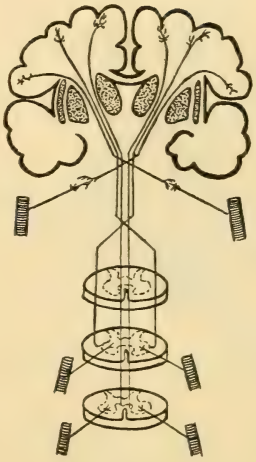


Fig. 203.—The direct (voluntary) motor tract. (After Van Gehuchten.)

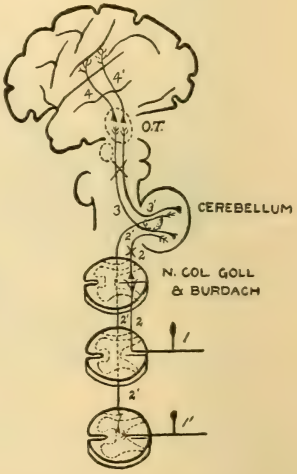


Fig. 204.—Indirect sensory tract. (After Van Gehuchten.)

Direct cerebellar.

the trophic influence of the anterior horn cells. The *direct cerebellar tract* represents chiefly visceral sensation and is concerned with

equilibrium. The *antero-lateral ascending tract* (Gower's tract) is concerned with sensations of pain, temperature and touch, conveyed from the opposite side through the anterior commissure. *Goll's tract* conducts sensation from the joints, tendons and muscles *of the same side*, while in the cord, *its fibres crossing in the medulla*, and it is therefore associated with ataxia, deficient orientation and muscle sense in general, under conditions of disease. *Burdach's tract* conveys tactile sensations from the opposite side, contains many associating fibres and is crossed by afferent sensory fibres conveying reflex, painful, articular or muscular

Gower's tract.

Goll's tract.

Burdach's tract.

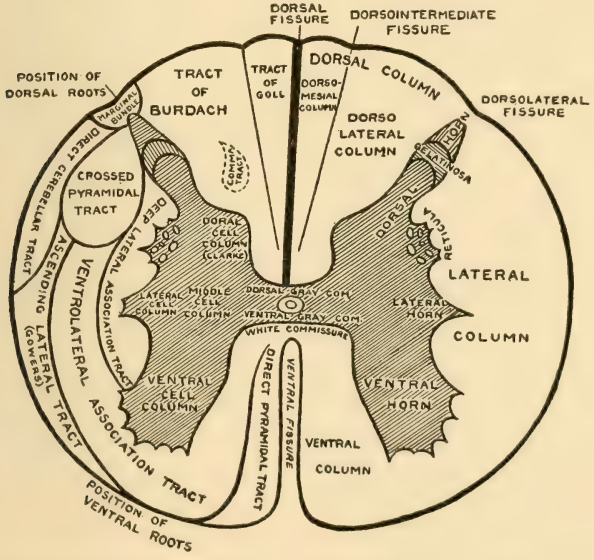


Fig. 205.—Diagrammatic transverse section of spinal cord. (After Gerrish.)

stimuli, hence if diseased there may be anæsthesia, ataxia, more or less pain and interrupted (lost) reflexes. *The fundamental columns* are related to association of different cord levels with each other and with the brain stem and cortex and contain both sensory and motor fibres. The cells of the anterior horns are trophic in function, constitute the nutrient cells of the lower motor neurones, and if diseased produce atrophy, R.D., and paralysis, (flaccid paralysis) and loss of the deep reflexes. The anterior horns are essentially motor, the posterior sensory. The cells at the posterior angle of the posterior commissure are probably

Fundamental columns.

Anterior horn cells.

Anterior vs. Posterior horns.

Automatic centres.

automatic centres, others in the immediate neighborhood may be trophic, sensory and vasomotor.

Significance of Tract Involvement in Disease.—In locomotor ataxia and Friedreich's ataxia the *posterior columns* are chiefly involved, in combined sclerosis, *both these and the lateral pyramidal tracts;* in lateral sclerosis the *crossed pyramidal tract* is chiefly affected; in amyotrophic lateral sclerosis both the anterior horns and the crossed pyramidal tract, while in anterior poliomyelitis and progressive muscular atrophy the anterior horns are chiefly involved.

Terms in Common Use.—*Neurosis.*—A morbid nervous state.* *Psychosis.*—Morbid mental state. *Functional Disease.*—One which lacks demonstrable anatomic changes. *Tremor, Spasm, Convulsions.*—See page 30. *Paralysis.*—Loss of motor power. *Paresis.*—Impaired motor power. *Monoplegia.*—Paralysis of one limb. *Hemiplegia.*—Paralysis of one-half the body. *Paraplegia.*—Usually used to indicate bilateral paralysis of the lower portion of the body. *Diplegia.*—Double hæmiplegia. *Anæsthesia.*—Loss of tactile sense. *Analgesia.*—Loss of pain sense. *Thermo-anæsthesia.*—Loss of temperature sense. *Paræsthesia.*—Perverted sensations. *Allochiria.*—Sensory stimulation of one side referred to opposite side. *Transferred or Referred Pain.*—Irritation referred to a distant region, usually as pain, i.e., headache from uterine disease, knee pain in hip joint disease, etc. *Delayed Sensation.*—Time elapsing between sensory stimulus and its perception exceeds 1-10 of a second. *Hyperæsthesia.*—Excessive sensibility. *Hyperalgesia.*—Excessive sensibility to pain. *Ataxia.*—Loss of voluntary co-ordinate movements. *Motor Ataxia.*—Loss of co-ordination in limb movements. *Static Ataxia.*—Loss of equilibration. *Cerebellar Ataxia.*—Loss of equilibration with a peculiar drunken gait unassociated with true motor ataxia.

Etiologic Factors.—In a book of this character but a slight attempt need be made to cover the anatomy and physiology of the nervous system. On the other hand a discussion of the etiology and general laws of pathologic change and the symptoms produced by lesions affecting different centres or conduction paths must be taken up in direct relation to general symptomatology.

Causes may be both *direct* or *indirect, primary* or *secondary,* and though the greater number of nervous diseases proper are distinctly related to structural alterations of the nervous system, the clinician

* Neuroses may be motor or sensory, trophic or secretory, vasomotor or thermic, or, a combination of these forms.

must always hold in mind the fact that remote disease may find its ultimate and perhaps chief and terminal expression in the brain, cord or peripheral nerve. For example, *arterio-sclerosis* is directly responsible for most cases of cerebral hemorrhage and thrombosis, as is *left-sided valvulitis* for embolus. *A peripheral neuritis* may be traced to *lead* or *alcohol* poisoning, and in such cases, reasoning from effect to specific cause, one reaches a definite and potent therapeusis. We may consider therefore without separating primary from secondary lesions the following etiologic factors.

Remote ailments as causes.

(a). *Toxæmias. Arsenic, lead, alcohol and carbon bisulphide* serve as the type of one form; *diphtheria, erysipelas, syphilis and typhoid* of another; *nephritis, diabetes and chronic auto-intoxication, severe anæmias and the cachexias* yet another. In most instances such poisons produce changes in the peripheral nerves or the spinal cord, i.e. polyneuritis, acute poliomyelitis, Landry's paralysis, locomotor ataxia, etc., the peripheral nerves being most often affected. (b). *Secondary* (metastatic) *tuberculous, malignant or septic foci.* (c). *Affections of the heart and blood vessels*, i.e. valvular lesions, arterio-sclerosis, degenerative diseases of the heart muscles, etc. (d). *Tumors.* Under this we include gummata, sarcoma, carcinomata, tuberculous growths, cysticus cysts, etc. (e). *Traumatism and its sequences* including scar formation. (f). *Meningeal inflammation*, acute or chronic, suppurative or non-suppurative. (g). *Caries, gumma, osteomyelitis.* (h). *Developmental defects and diseases of a distinctly hereditary type.* (i). *Certain unexplained nervous disorders* denominated functional, such as epilepsy and chorea. (j). *Certain changes of a purely senile sort*, often premature in occurrence, and in general, degenerative and atrophic in type. (k). *Hereditary* and *acquired predisposition, habits* and *environment*, hold a prominent place.

Degeneration.—Injury to the *parent cell causes degeneration of its processes*, and *division of these leads to their death peripherally, but may not entirely destroy the function of the cell.* It is interesting to note that secondary degeneration is limited to the neurone involved and that a lesser amount of atrophy may occur in a fibre not directly or exclusively connected with the affected cell.

The cell and its process.

There are three chief forms of degeneration.—(1). *Primary.* (2). *Secondary or Wallerian.* (3). *Toxic.* The *first* occurs in the cachexias, locomotor ataxia and in senile processes. The *second* is characterized by a degeneration chiefly affecting the peripheral segment and is a relatively rapid process, being complete within from 2–4 weeks. As

Three types.

a rule the degeneration follows the direction of normal impulse conduction, though in *association* or *commissural* fibres it is only partial and as the *trophic centre of the sensory nerve is the spinal ganglion*, if the injury or section be *between the ganglion and the cord* the peripheral

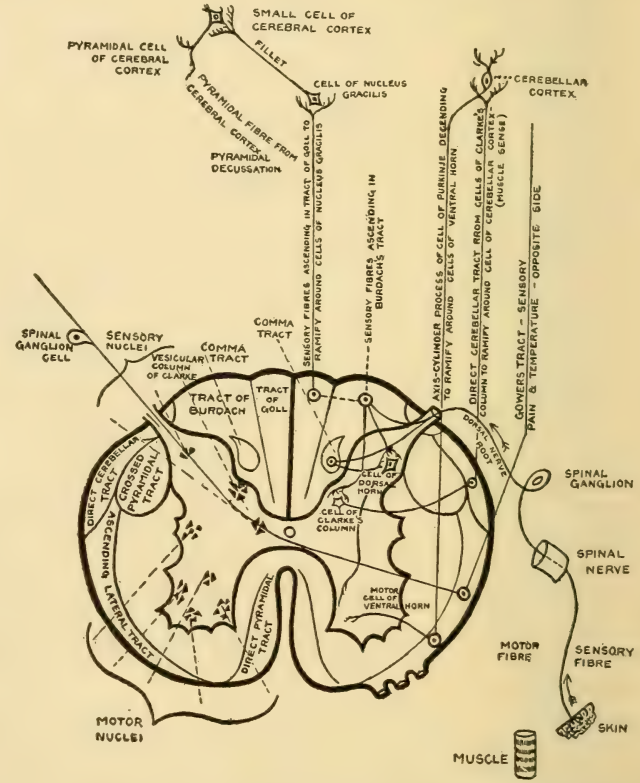

SMALL CELL OF CEREBRAL CORTEX
PYRAMIDAL CELL OF CEREBRAL CORTEX
FILLET
CELL OF NUCLEUS GRACILIS
CEREBELLAR CORTEX
PYRAMIDAL FIBRE FROM CEREBRAL CORTEX
PYRAMIDAL DECUSSATION
SENSORY FIBRES ASCENDING IN TRACT OF GOLL TO RAMIFY AROUND CELLS OF NUCLEUS GRACILIS
SENSORY FIBRES ASCENDING IN BURDACH'S TRACT
AXIS-CYLINDER PROCESS OF CELL OF PURKINJE DESCENDING TO RAMIFY AROUND CELLS OF VENTRAL HORN
DIRECT CEREBELLAR TRACT FROM CELLS OF CLARKE'S COLUMN TO RAMIFY AROUND CELL OF CEREBELLAR CORTEX (MUSCLE SENSE)
GOWER'S TRACT - SENSORY PAIN & TEMPERATURE - OPPOSITE SIDE
SPINAL GANGLION CELL
SENSORY NUCLEI
VESICULAR COLUMN OF CLARKE
COMMA TRACT
COMMA TRACT
TRACT OF BURDACH
TRACT OF GOLL
DORSAL ROOT
CELL OF DORSAL HORN
CELL OF CLARKE'S COLUMN
SPINAL GANGLION
SPINAL NERVE
DIRECT CEREBELLAR TRACT
CROSSED PYRAMIDAL TRACT
ASCENDING
LATERAL TRACT
DIRECT PYRAMIDAL TRACT
MOTOR CELL OF VENTRAL HORN
MOTOR FIBRE
SENSORY FIBRE
MOTOR NUCLEI
SKIN
MUSCLE

Fig. 206.—Showing course of sensory and motor fibres in the cord and the formation of a spinal nerve. (After Keiller, Gerrish and Dana. Slightly modified.)

nerves do not suffer. The *cells of the anterior horns are trophic for motor nerves* and injury to them as to the peripheral nerve itself means outwardly progressing degeneration. The *third*, toxic degeneration, is segmental but follows the laws of Wallerian degeneration. *Regeneration.* Nerve cells cannot be reproduced if destroyed, but nerve fibres

can regenerate though only as to the peripheral fibres, and this process, moreover, demands that the trophic centre be sound.

Classification.—Whatever be the cause of organic disease of the nervous system, we distinguish those of general or irregular distribution from those which chiefly or perhaps exclusively affect a definite tract or neuronic chain ("system disease"). In "*focal*" disease a certain portion of the brain or spinal cord may be involved together with all contained structures. If two or more definite conduction paths are involved one speaks of a "*combined system*" disease. The distinction cannot be exact but is convenient and conforms to known disease types. Anterior poliomyelitis, primarily a system disease, may be followed by a diffuse secondary sclerosis. In the grosser lesions such as those of hemorrhage, thrombosis and abscess, one finds a tendency to more or less rapid necrosis of tissue resulting in cyst formation, which may show gradual occlusion by contraction and scar formation.

Sequence of Degenerative Changes.—These may be thus summarized*:—*Spinal Cord Lesions.*—Ascending degeneration of sensory pathways, descending degeneration of motor tracts. *Region of the Mesencephalon.*—Degeneration of the optic tracts and red nucleus, atrophy of the brachia, corpus dentatum of the cerebellum, pontine ganglia, etc., and both ascending and descending degeneration of the fillet. *Cerebellar Lesions.*—Atrophy of the opposite red nucleus and the brachia; degeneration of the median cerebellar peduncle, restiform body, lower olive, etc. *Cortical Lesions.*—Atrophy of such related structures as the red nucleus, fillet, opposite brachium, and the subthalamic region with degeneration of the optic thalamus, all directly related projection fibres and partial degeneration of those of association. In young children Wallerian degeneration results in arrested growth of the parts involved as is well illustrated by those adults who have had infantile spinal palsy.

General Relation of Pathologic Changes to Symptomatology.—The following summary will suffice to show the direct and indirect results of (a) *irritation*, or (b) *destruction* of the motor and sensory cells and pathways. In the former instance we find morbid activity, in the latter loss of function.

A sharp contrast.

Sensory Centres and Pathways.—*Irritation.* (a). *Increased response to peripheral stimuli*, i.e. hyperæsthesia or actual pain (hyperalgesia, violent neuralgic pains). (b). *Subjective sensations* such as

Excessive response.

* According to Jakob.

tinnitus aurium, hallucinations, both visual and aural, and various paræsthesias such as numbness, tingling, formication, etc. *Destruction.* Absent response. Loss of sensory response to peripheral stimuli wholly or partly as to all or only certain special sensations (i.e. anæsthesia, thermo-anæsthesia, analgesia, etc.), partial or complete ataxia.

Girdle Sensation.—In locomotor ataxia, chronic myelitis and certain cases of tumor or injury to the spinal cord or its meninges, a subjective sensation of constriction of the trunk may be manifest quite similar to that experienced after violent or prolonged fits of coughing, vomiting or sneezing.

Motor Lesions.—(A). *Peripheral or lower motor neurone* (i.e. anterior horn cells, or cranial nuclei, to periphery). *Paralysis is flaccid, usually bilateral** (if unilateral on same side as lesion), and *must* be due to a *lesion involving* (a) *the cells of the anterior cornua*, (b) the *cranial nuclei*, or, (c) the *peripheral nerves*, cranial or spinal as the case may be. *Fibrillary* twitching may be present but there is no *contracture* or *rigidity*, and by interrupting both the reflex arc and the trophic stimulus it *involves a loss of the reflexes* and the supervention of *marked muscular atrophy* and the *electric reaction of degeneration.* Not only does the *presence* of reaction of degeneration conclusively *prove such a paralysis to be one of the peripheral neurone*, but its *entire absence* after several days means early recovery. *In general, the prognosis is good in inverse proportion to the degree of R. D. impairment.*

Flaccid paralysis. Site of lesion. Lost reflexes and atrophy. R. D. Prognosis.

(B). *Central Motor Neurone* (i.e. cortical centres to cranial nuclei or anterior horn cell but not including them). On the side *opposite an intracranial lesion* there is *spastic paralysis* (rigidity) with *secondary contractures*, whereas *marked true atrophy* (aside from that of disuse) *is lacking, R. D. absent* and *tendon reflexes increased* by removal of cerebral inhibition. **Irritative lesions affecting motor areas of the cortex** may cause *tonic, clonic* or *epileptiform* spasms, *athetoid, choreic or wholly irregular movements and muscular twitching.*

Spastic type. No R. D. or atrophy. Reflexes increased. Cortex irritation.

Unilateral Spinal Cord Lesions.—(Brown-Sequard paralysis). Loss of power is complete on the side of the lesion with anæsthesia on the opposite side save for a *zone* corresponding to the level of the lesion *and on the same side.* Muscle sense is *impaired on the paralyzed side*, retained *on the opposite* (*anæsthetic*) *side*, and the type of paralysis is *spastic* save at the level of the lesion where the destruction of the anterior

Hemiparaplegia.

* Cord lesions are usually *bilateral;* cortical lesions *crossed monoplegic;* lesions of the region below the cortex and above the motor decussation in medulla, *crossed hemiplegic.*

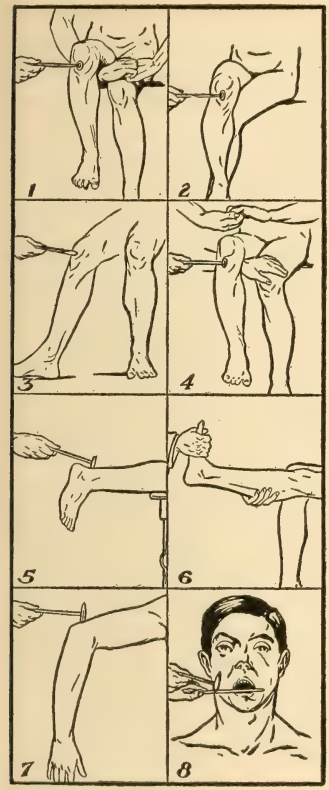


Fig. 207.

1. Knee jerk elicitation. Method employed by author.
2. Simple method.
3. A common method.
4. Ordinary re-enforcement method.
5. Method of obtaining Achilles jerk.
6. Method of eliciting ankle clonus.
7. Method of obtaining triceps jerk.
8. Method of obtaining jaw jerk.

horn cells may produce a *flaccid paralysis in the limited area represented* by their trophic influence.*

Complete Transverse Spinal Cord Lesions.—Symptoms.—If the transverse lesion be complete there is complete *flaccid paralysis* and anæsthesia below the lesion with *lost deep reflexes, atrophy,* and *R. D.* Any spastic paralysis indicates an incomplete lesion.

Flaccidity and anæsthesia.

THE REFLEXES.†

Cilio-spinal Reflex.—Stimulation of the cervical sympathetic by pinching the skin of the neck dilates the pupil and its absence indicates a lesion of that nerve.

Conjunctival Reflex.—Complete anæsthesia, deep stupor, or coma usually abolishes the well known spasm of the orbicularis palpebrarum following conjunctival irritation.**

Palate and Pharynx Reflex.—The latter is absent in most hysterias.

Jaw Jerk.—Percuss the chin firmly by the mediate method while the mouth is moderately open. (A *contraction of the muscles of mastication.*) Inconstant in health, marked if reflex excitability is increased.

Supinator Jerk.—Tap supinator tendon just above styloid insertion. Usually absent in health (*5th cervical segment*).

* The relatively prompt crossing of the sensory paths other than those of muscle sense explains the anæsthesia of the non-paralyzed side and the interruption of the sensory root fibres at the level of the lesion accounts for the *zone* of anæsthesia upon that side, above which is a band of hyperæsthesia.

† In general the presence of normal *superficial* reflexes is much more important than their absence.

** Hysteric anæsthesia also.

Triceps Jerk.—Support the arm at the elbow carrying it outward at a right angle to the body, let the forearm hang vertically downward and tap strongly the triceps tendon just above the olecranon (*5th, 6th, and 7th cervical segments*). It is absent or slight in health.

Biceps Jerk.—Flex elbow at right angles, strike forearm near lower end of radius. Biceps contracting occurs only in disease (*5th cervical segment*).

Achilles Jerk.—Put the tendon Achilles on the stretch by extending the leg and dorsiflexing the foot, or have the patient kneel on a chair with the foot unsupported; tap it sharply and in health a contraction of the calf muscles follows. Its early disappearance in locomotor ataxia is an important diagnostic sign (*5th lumbar and 1st sacral segment*).

May precede loss of knee jerk.

Scapular Reflex.—Contraction of scapular muscles on stroking interscapular region (*5th, 6th, 7th and 8th cervical and 1st dorsal segments*).

The Patellar Reflex.*—(Knee jerk). This is best obtained by having the patient cross one knee over the other or better by passing his own arm over the opposite knee and under the knee of the member to be tested, so that the leg hangs *free and relaxed* across the wrist. Then the patient is told to close the eyes, join his own hands and *pull when the word is given* (re-enforcement), *coincidently the tendon just below the patellar margin is sharply tapped* with the side of the hand, the edge of a book or a percussion hammer.† The forward jerk of the leg and foot or visible contraction of the muscle varies greatly in health and may be markedly increased in neurasthenia, hysteria, tetanus, strychnine poisoning, rheumatoid arthritis or after sexual excesses and drinking bouts, *and is pathologically increased in any disease which cuts off the cerebral inhibition fibres.* Such are *cortical* hæmiplegias and *sclerosis of the lateral columns* (lateral or amyotrophic). For clinical purposes one may assume that it is invariably present in health and *its absence indicates a lesion interrupting the reflex arc*, viz., a lesion involving the sensory or motor fibres (neuritis), the posterior roots or columns, the anterior cells or even the motorial end plates. *Hence it is lost in locomotor ataxia, anterior polio-*

Re-enforcement.

Increased knee jerks.

Lost knee jerk.

* *Tendon Reflexes.* (Deep reflexes.) It will be noted that all of the tendon reflexes require that the tendon involved be *slightly* on the stretch.

† Usually the jerk can be obtained without re-enforcement, but it usually saves time to carry out the entire procedure. In bed-ridden patients the same procedure can be carried out by utilizing one's own arm and the flexed opposite knee as points of support.

myelitis, transverse myelitis of the 2nd and 3rd lumbar segments (seat of reflex), *Landry's paralysis, Friedreich's ataxia* and sometimes in chorea, diabetes and severe toxæmias such as diphtheria. Cases of pathologic exaggeration yield the "jerk" on tapping the *quadriceps tendon* above the patella. In such cases especially "*ankle clonus*" should be sought.*

Ankle Clonus.—*The knee is very slightly flexed, the heel rests in the palm of the examiner's left hand, his right grasps, extends and suddenly dorsiflexes the foot upon the leg. An initial series of clonic involuntary contractions of the muscles of the calf repeated under sustained pressure of the flexing hand constitutes clonus.* True clonus has the same significance as exaggerated knee jerks in its relation to *organic* disease, being most common in lateral and disseminated sclerosis. If contractions appear before the degree of foot flexion exceeds a right angle and are evidently voluntary, irregular and fleeting, one is dealing with spurious clonus, usually hysterical. (*2nd and 3d lumbar segments.*) Tonic contraction of the anterior tibial group may occur.

True clonus prolonged.

Pseudo-clonus, transient, irregular and voluntary.

Wrist Jerk.—Let the hand drop at the wrist (which is supported), strike the extensor tendons of the dorsum just proximal to the wrist joint. (Usually absent or slight in health.)

Epigastric.—Retraction of epigastrium following downward stroking of chest in nipple line. A continuation of this line of stroking from the costal margin downwards produces contraction of the abdominal muscles. It is of little importance or clinical value. (*4th, 5th, 6th and 7th dorsal segments.*)

Abdominal Reflex.—Elicited by stroking outer side of abdomen at various levels (*8th, 9th, 10th, 11th and 12th dorsal and 1st lumbar segment*).

Gluteal Reflex.—This consists in contraction of the glutei on vertical stroking of the buttocks (*4th and 5th lumbar segments*).

Cremasteric Reflex.—Quick retraction of testicle upon stroking inner and upper part of thigh. (Not dartos contraction.)

Caution.—All reflexes are best elicited when the patient's attention is diverted. The superficial reflexes merely prove the integrity of the region governing them.

Babinski's Toe Reflex.—This consists in *deliberate* dorso-extension and separation of the toes and especially of the great toe, followed by dorsi-flexion of the ankle joint, when the sole is gently or firmly

*The *real test* symptom is contraction of the quadriceps muscle rather than actual movement of the foot. Hence the examiner's free hand should be placed upon the muscle.

stroked with a quill point, finger nail, etc. It is usually associated with disease involving the *pyramidal tracts* and in any event is pathologic. It is said that in healthy young children not yet able to walk a similar but *more rapid* response is obtainable.

Plantar Reflex.—*The chief value of this sign lies in its constancy in health and frequent absence in hysteria.* If the sole of the foot, thoroughly dried, be stroked, the patient's attention being diverted, his position supine, the knee and thigh semiflexed with the leg in outward rotation, plantar flexion of the toes beginning with the four outer, and dorsi-flexion of the ankle result (*2d and 3d sacral segments*).

Fig. 208.—Plantar reflex. Upper plate shows normal reflex plantar flexion. Lower:—Dorsi-flexion of great toe. (Babinsky's sign.)

Oppenheim's Reflex.—This is a Babinsky reaction elicited by forcibly stroking ths inner border of the tibia. Gordon applies deep pressure to the calf muscles.

Adductor Jerk.—Abduct thigh and tap adductor magnus tendon. Adductor contraction may occur on both sides (crossed adductor jerk) in conditions of high reflex irritability and under the same conditions one may obtain **knee clonus** (clonic contractions of quadriceps). Extend leg and push patella sharply to the front, maintaining pressure for a considerable period.

Organic Reflexes.—These involve chiefly respiration, deglutition, micturition and defecation, and a full consideration of their complicated mechanism is out of the question in a volume on medical diagnosis, and are best considered in relation to the symptomatology of the individual diseases.

Defecation.—To test the action of the rectal sphincter a digital examination is necessary, the strength of the resistant or grasping contraction being noted. In health contraction follows a prick of the anal region. Rectal incontinence may be met with in all conditions associated with coma or profound toxæmia such as typhoid, as well as in certain organic nervous diseases. There is sometimes a true reflex spasmodic incontinence.

Micturition.—Dribbling of urine usually means overfilling of the bladder and calls for the use of the catheter; a true reflex incontinence

may exist such as is seen as a temporary phase in young children. Both rectal and vesical centres are in the lower lumbar (*4th and 5th*) and upper sacral (*1st, 2d, 3d portion of the cord*).

THE EXAMINATION OF THE MUSCLES.—The tests of paralysis are suggested by the defects noted under segmental lesions, page 543, or under discussion of the special nerves involved. A *peculiar lack of tone is noticeable in a paralyzed limb even though the patient be unconscious* and muscular atrophy is readily detected by observation, careful measurement and palpation. With this should be considered the question of power and electrical reactions as in certain diseases a spurious hypertrophy appears.

and nerve lesions.

Lack of tone.

Loss of power.

Electrical Tests.—*The reaction of degeneration.* Faradic and galvanic batteries are required together with the usual appliances for controlling and applying the current. *The muscles should first be tested for faradic excitability.* Having wet both electrodes with salt water a weak current is applied, and, with the indifferent electrode over the sternum or between the scapulæ, *the smaller electrode is applied successively to the motor points of Erb and the resulting tonic muscular contraction and the increase of current necessary to produce it is noticed.* Corresponding points on opposite sides of the body should be tested in alternation. *The galvanic current. The indifferent electrode* (the larger) *should be positive, normal electrode negative.* These are applied as in the faradic test *and the current is first closed and after a moment again opened, gradually increasing the current until muscular contraction follows the closure of the circuit* this constituting cathodal closure contraction (CaCC). *The switch is then thrown to change the direction of the current, the intensity of which must remain the same* and the test repeated when *muscular contraction will represent anodal closure contraction* (ACC). The *comparative* promptness and strength of contraction must be carefully noted. Further tests are not necessary but the opening contractions may be noted in the same manner.

R. D.

Faradic test.

Galvanic test.

Cathodal closure contraction.

Anodal closure contraction.

Significance of Electrical Reactions.—Normally the response should be prompt and decided to either electrode of the *faradic current and should continue during its passage. If the contraction be sluggish, or absent, if it diminishes or disappears, it indicates disease.* So also in the case of the *galvanic current, the reaction following cathodal closure* should *occur with a less current than is required for anodal closure* contraction but no tonic contraction should occur while the current is passing. Similarly AnCC exceeds AnOC and this in turn CaOC. *The reaction of degeneration, however, is represented by an AnCC which equals or*

Promptness of reaction.

CaCC > AnCC is normal.

Sluggish response.

exceeds CaCC. Furthermore, in the case of the galvanic as in the faradic, contraction may be delayed, sluggish or vermiform or be entirely absent. R.D. *signifies a loss of nutrition in the motor nerve due either to disease* of a trophic centre or the nerve itself *and is a promi-*

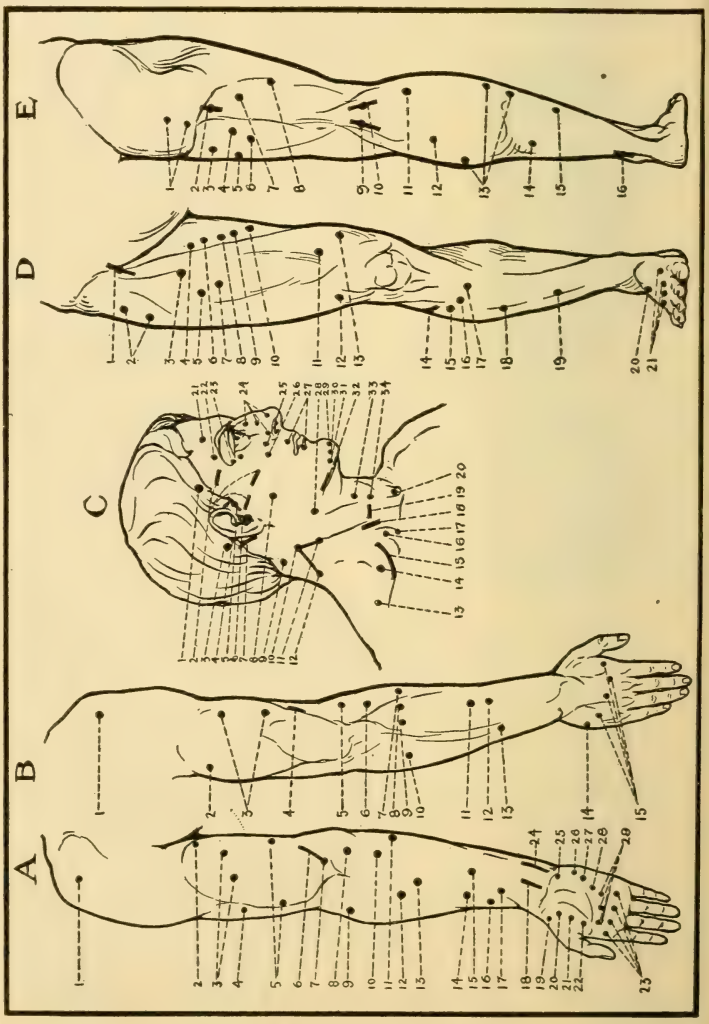


Fig. 209.—The motor points of Erb.

A. Anterior surface of arm, shoulder and hand.

1. Deltoid.
2. Musculo-cutaneous n.
3. Biceps.
4. Musculo-spiral n.
5. Brachialis anticus.
6 & 7. Median (the ulnar lies just internal to 6).
8. Pronator radii teres.
9. Supinator longus.
10. Palmaris longus.
11. Flexor carpi ulnaris.
12. Flexor carpi radialis.
13. Flexor profundus digitorum.
14. Flexor sublimis digitorum.
15. Same (2nd and 3rd).
16. Flexor longus pollicis.
17. Pronator quadratus.
18. Median n.
19. Abductor pollicis.
20. Opponens pollicis.
21. Flexor brevis pollicis.
22. Adductor pollicis.
23. Lumbricales.
24. Ulnar n.
25. Palmaris brevis.
26. Adductor minimi digiti.
27. Flexor brevis minimi digiti.
28. Opponens minimi digiti.
29. Palmar interossei.

B. Posterior surface of arm, shoulder and hand.

1. Deltoid.
2. Triceps (long head).
3. Triceps (external head).
4. Musculo-spiral nerve.
5. Supinator longus.
6. Extensor carpi radialis longior.
7. Extensor carpi radialis brevior.
8. Extensor communis digitorum.
9. Supinator brevis.
10. Extensor carpi ulnaris.
11. Extensor indicis.
12. Abductor longus pollicis and extensor brevis pollicis.
13. Extensor longus pollicis.
14. Abductor minimi digiti.
15. Dorsal interossei.

C. Motor points on the head and neck.

1. Temporalis.
2. Middle branch. (Facial) two lower (branch of upper above).
3. Occipitalis.
4. Retrahens aurem.
5. Upper facial.
6. Facial trunk.
7. Posterior auricular nerve.
8. Masseter.
9. Splenius.
10. Spinal accessory nerve.
11. Sternocleidomastoid.
12. Trapezius.
13. Long thoracic nerve (serratus magnus).
14. Circumflex nerve (deltoid).
15. Brachial plexus.
16. Erb's point (deltoid, biceps, brachialis anticus, supinator longus).
17. Anterior thoracic nerve (pectoralis major).
18. Phrenic nerve.
19. Omohyoid.
20. Sternothyroid.
21. Frontalis.
22. Corrugator supercilii.
23. Orbicularis palpebrarum.
24. Nasal muscles.
25. Levator labii superioris.
26. Zygomaticus major.
27. Orbicularis oris.
28. Hypoglossal nerve.
29. Levator labii inferioris.
30. Depressor labii inferioris.
31. Depressor anguli oris.
32. Lower branch of facial.
33. Platysma.
34. Sternohyoid.

D. Motor points on the anterior aspect of the leg.

1. Anterior crural nerve.
2. Tensor fasciæ latæ.
3. Sartorius.
4. Obturator nerve.
5. Quadriceps (common point).
6. Pectineus.
7. Rectus femoris.
8. Adductor longus.
9. Adductor magnus.
10. Gracilis.
11. Crureus.
12. Vastus externus.
13. Vastus internus.
14. External popliteal nerve.
15. Peroneus longus.
16. Extensor longus digitorum.
17. Tibialis anticus.
18. Peroneus brevis.
19. Extensor hallucis longus.
20. Extensor brevis digitorum.
21. Dorsal interossei.

E. Motor points on the posterior aspect of the leg.

1. Gluteus maximus.
2. Sciatic nerve.
3. Adductor magnus.
4. Semitendinosus.
5. Gracilis.
6. Semimembranosus.
7. Biceps (long head).
8. Biceps (short head).
9. Internal popliteal nerve.
10. External popliteal nerve.
11. Gastrocnemius (outer head).
12. Gastrocnemius (inner head).
13. Soleus.
14. Flexor longus digitorum.
15. Flexor longus hallucis.
16. Posterior tibial nerve.

Significance of R. D.

nent symptom of the flaccid palsies whether affecting cranial or spinal nerves. *When R. D. is present the faradic excitability is lost.*

CERTAIN PSYCHIC AND SENSORY DERANGEMENTS.

Tests.

Defective Memory and Mental Obtuseness.—A patient's memory should be noted in the preliminaries of case-taking, and he may be specially questioned in regard to what he has had for dinner or breakfast, his birthday, the number and ages of children, etc. Mere dulness or confusion of ideas such as is associated with a multitude of disease conditions will of course obscure memory, but should be ascribed to their true cause. In some instances it may be due to organic lesions of the brain, such as apoplexy, tumor, thrombosis, embolism, meningitis, senile degeneration or sclerosis, or true insanity, or on the other hand to mere anæmia or commonly to neurasthenia. Actual loss of memory is chiefly observed in the true insanities, chronic alcoholism and neurasthenia.

Discrimination required.

Grave or trivial.

Illusions, Delusions and Hallucinations.—An *illusion* is a faulty interpretation of an actual visual impression and is actually a visual paræsthesia common to nearly all forms of delirium; a babe may appear an ogre, a poker a serpent, etc.

Delusions are false and irrational beliefs, unsystematized in delirium and systematized in insanity. **Hallucinations** are sensory impressions lacking any objective basis, and it will be readily seen that these various mental derangements are mutually contributory.

Active vs. Passive.

Delirium.—Is in itself divisible into active (wild or maniacal), passive (low, muttering). Furthermore, a delirium may be wholly nocturnal and in all acute diseases tends to assume that form or merge gradually into it during convalescence. Delirium tremens and the fully developed cases of typhoid fever illustrate respectively the active and passive type. Delirium aside from brain lesions indicates chiefly toxæmia or extreme exhaustion, but may be seen in hysteria and occasionally in epilepsy.

Causes.

Disturbances of Sleep.—Insomnia may occur in acute and chronic diseases, whether cerebral or general, or in neurasthenia or simple overwork. Early waking or frequent waking is quite general in old persons, night terrors and transient somnambulism are common in young children. *Tea, coffee, tobacco, cerebral excitement, old age, and fatigue* are the common causes of sleeplessness.

Disturbances of Speech.*—*Normal speech depends upon the normal*

*See also p. 29.

interaction of the senses of sight, touch, smell, taste, sound and muscle sense, together with intact association fibres, a normal memory and an intact motor mechanism. If any link in this chain be lacking a defect in speech results, often taking the form which corresponds to the particular function or subfunction which is deficient. Thus *anarthria or dysarthria* may result from a defective motor tract or from the lack of power to perform the co-ordinate movement necessary to word formation. This is characterized by imperfect articulation of the linguals l and t if the tongue be involved, or of the labials b, p and n which are replaced by f and v if the lips are affected. Palatal paralysis causes explosive p's and b's and makes the voice itself nasal.

Complex association.

Anarthria.

Changes in Rhythm.—*Syllabic speech* (division of words into syllables), *scanning and staccato speech* (explosive word utterance) is another form frequently observed in multiple sclerosis and occasionally in other conditions. Opposed to this is the *confluent speech* usually bulbar in origin. In general one may say that *lesions involving the medulla and pons give rise to anarthria or dysarthria, whereas cerebral lesions cause speech defects of an entirely different type in which the perceptive centres and higher and more complex degrees of co-ordination are involved.* The lower mechanism is of course dependent upon and subservient to the higher.

Syllabic scanning staccato.

Confluent.

Causative lesions.

Disturbances of Speech of Cerebral Origin.—Such are included under the general term *aphasia* which is divided into *motor and sensory types*, the former representing the higher emissive mechanism, the latter the power of receiving and reviving speech concepts, being largely dependent upon sight and hearing (auditory and visual centres). The motor speech centre (Broca's centre) in the inferior third left frontal convolution and the articulation centre in the inferior anterior central convolution govern motor speech and either may be affected independently of the other. A lesion of the left motor speech centre causes entire loss of voluntary speech, i.e. true motor aphasia, but does not affect automatic and emotional speech, the centre for which exists in both hemispheres. It is evident that motor aphasia logically includes the loss of power to utter given words (aphemia), to write words (agraphia), to use gestures (amimia) and the loss of power of musical expression, vocal or written (motor amusia and musical agraphia).*

Motor vs Sensory.

Broca's centre (on opposite side in left handed persons).

Automatic centres.

Amimia and agraphia.

* Ataxic aphasia covers error in form without articulation defects or any evidence of sensory aphasia beyond slight or marked errors in the pronunciation of words, and is a form of aphasia usually seen in attacks that are partial or during the process of recovery. The term is sometimes applied to motor aphasia in general.

Word deafness and word blindness.

In sensory aphasia the inability to *perceive* and *interpret* language is fundamental and therefore there must be an *auditory aphasia* or word deafness (loss of power to recognize spoken words), *visual aphasia* or alexia (failure to recognize or understand written or printed words, word blindness), and subdivisions of exactly the same sort as were referred to under motor aphasia. In *paraphasia*, word repetition, misuse and incoherence are the chief features and *paragraphia* represents the graphic form of the same subdivision.

Paraphasia and paragraphia.

Mind blindness and mind deafness represent an entire loss of visual or auditory memory, not only of words but of common objects and the faces even of near relatives, or in the second variety all power to recognize musical or other sounds disappears. Here again we find subdivisions such as *apraxia* or inability to recognize the use of objects, and *alexia* representing a failure of comprehension in reading.* *Amnesic aphasia* expresses the difficulty in recalling *voluntarily* special words or classes of words.

Apraxia and alexia.

Amnesic aphasia.

The Lesions Affecting Speech.—*Motor aphasia*—third left frontal convolution in the right handed or a subcortical lesion underlying Broca's centre. *Visual speech*—left angular and supramarginal convolutions. *Auditory aphasia*—first temporal convolution, auditory speech centre or afferent tracts.

Localization.

Lesions of the association fibres are of course frequently combined with others as in *apraxia*. *Anarthria* may of course result from lesions in any portion of the tract interrupting stimuli from the higher mechanism or affecting the centres in the medulla and pons or indeed the cranial nerves themselves.

Method of Examination.—(a). *Motor aphasia* (aphemia), patient cannot speak voluntarily, read aloud or repeat words. If he cannot write voluntarily or by dictation or copy he has *agraphia*. If in speaking or writing he uses wrong words or is strikingly incoherent there is paraphasia or paragraphia. (b). *Sensory aphasia*. The patient cannot recognize words written or printed and read them *silently* even, or he cannot read them intelligently or correctly. This is determined by writing direct simple questions to be answered by the patient. If he cannot hear spoken words or understand them as indicated by his failure to perform some ordinary action at command he has *auditory aphasia*.

Tests.

* Another rare subdivision known as *dislexia* covers a peculiar mental fatigue accompanying reading and even for short periods unassociated with visual defects or pain.

Comment.—In testing for both visual and auditory aphasia the sight and hearing should be previously tested. Caution.

Mind Blindness and Mind Deafness.—For testing the former one should use common objects such as a pencil, pen, pocket knife or coin and ask the patient to use them, i.e. to write, to open the knife, to open a book, etc. Mind deafness should be tested by the slamming of a door, ringing a bell, coughing, etc., etc. Tests.

INVESTIGATIONS OF SENSORY FUNCTIONS.—The eyes of the patient should be bandaged and one should test, 1st. *General sensation.* 2nd. *Pain.* 3rd. *Temperature sense.* 4th. *Muscle sense.*

Touch.—The tip of a pen, quill, toothpick or pencil point and a lightly rolled bit of cotton, a feather or a camel's hair brush are necessary for determining variations in sensibility, the latter articles serving to eliminate the element of pressure. The patient must be instructed to immediately say "touch" when any stimulus is perceived and he must be frequently checked by omitting the touch though putting the question. Exactly similar areas on both sides should be systematically followed to avoid bungling and time consuming repetition. He should also be made to indicate with his finger the exact points tested, or distinguish between normal, diminished (*hypæsthesia*), absent (*anæsthesia*), and excessive (*hyperæsthesia*) response. Such changes may be unilateral (*hemianæsthesia*), bilateral or even general or limited to a single member. As regards perception of stimuli they may be prompt or delayed, and finally we have to deal with various forms of perverted sensory perception (*paræsthesia*), amongst which are many of special sense and the so-called "*aura*" of epilepsy. *A failure to correctly localize sensations* is common in disease and subjective *transference* to the opposite side is called "*Allochiria.*" Testing. Be systematfc. Varieties of sensory defects. Delayed sensation. Paræsthesias. Referred sensation.

Tactile Sense.—This may be tested by the esthesiometer or lacking that an ordinary hairpin or pair of blunted or guarded compasses. In this both *pressure and contact* are included and the two points should be separated according to the part examined and in conformity to the following table* of normal perception, inasmuch as one point may be felt when two are applied or vice versa. The distances necessary for the sensation of two points in actual contact may be abnormally increased. In testing contact or touch the pressure sense may be determined by Simple appliances. Pressure sense.

*Lips. 3 mm. ($\frac{1}{8}$ inch). Tip of toes, cheeks, eyelids, temple. 12 mm. ($\frac{1}{2}$ inch). Tip of tongue. 1 mm. ($\frac{1}{25}$ inch). Tip of fingers. 2 mm. ($\frac{1}{12}$ inch). Tip of nose. 8 mm. ($\frac{1}{3}$ inch). Back of hands. 30 mm. ($1\frac{1}{4}$ inch). Forearm, leg, dorsum of foot. 40 mm. ($1\frac{3}{5}$ inch). Back. 60–80 mm. ($2\frac{2}{5}$–$3\frac{1}{5}$ inches.) Arm and thigh. 80 mm. ($3\frac{1}{5}$ inches).

Caution.

placing articles of different weight upon different portions of the body, always seeing that they are supported. *Localization* is readily tested by having the patient place his own finger upon any spot touched and the pain sense is determined by using a pen or pin. *Temperature sense* is tested most conveniently with test-tubes containing warm and cold water.* Any of the varieties of sensation above described may be increased and diminished, absent or misinterpreted.

Muscle Sense.—For proper testing, articles of various weight but of similar form should be used, the portion of the body tested being *unsupported. The tendinous or articular sense* is tested by having the patient imitate any movements of extension or flexion or unusual position in which the other leg is placed and further have him assume positions as described.

Ataxia.

Ataxia is chiefly dependent upon the articular and muscle sense but also involves sight, touch and other factors necessary to the harmonious action of muscle groups. It may affect either station, gait or other voluntary movements. *The Test.* The patient should be asked to touch the lobe of the ear of the opposite side, tip of the nose or a certain finger of the opposite hand or in the lower extremities touch different points of the opposite members of the great toe. He should then be required to stand with heels and toes together; *marked swaying or a fall* indicating static ataxia (Brauch Romberg sign). *If with the eyes open he cannot walk a straight line he has motor ataxia. If his gait is reeling and drunken* (titubating) *he has cerebellar ataxia* and in this form *the patient shows normal muscle sense and co-ordination if recumbent*, the difficulty being one of equilibration.

Static ataxia.

Motor ataxia.

Cerebellar ataxia.

Ordinary ataxia indicates cortical lesions or those of the pons crura and corpora quadrigemina, locomotor ataxia, transverse spinal lesions, ataxia, paraplegia, Friedreich's disease and syringomyelia. **Stereognosis.**—The inability to recognize familiar objects by touch (parietal lobe) usually suggests a lesion of the parietal lobe.

Significance of Sensory Disturbance.—*Hemianæsthesia of hysterical origin* commonly affects the left side, is sharply defined and frequently complete even as to the special senses. *Cortical hemianæsthesia* is combined with hemiplegia and usually incomplete unless associated with an unusually large lesion or involving the optic thalamus. *Crossed hemianæsthesia associated with hemiplegia of the opposite side* has been referred to as present in unilateral lesions of the cord (Brown-Sequard paralysis). *Hemianæsthesia and hemiplegia with*

Hysterical.

Cortical.

Unilateral spinal.

*Normally 60° F. is called cold and 85° F. warm.

crossed oculo-motor paralysis indicates a lesion of the *crus*, and finally, combined hæmianæsthesia and hæmiplegia *may* occur as the result of a lesion of the *internal capsule*. *Anæsthesias of patchy distribution suggest hysteria or neuritis* and *as between those mono-anæsthesias due to a spinal lesion and the rare cerebral form the former presents a sharp boundary line, the latter an anæsthesia diminishing as the trunk is approached.* As between *hysteria and neuritis the former tends to disregard both segmental and individual nerve distribution,* the latter is distinctly in accordance with them. *Bilateral anæsthesia* without motor paralysis usually affects the lower portion of the body and is particularly common in traumatic neuroses and hysteria. If the latter, *it spares the skin of the genitals and a portion of the sacrum,* if from the spinal cord it would almost invariably be associated with paralysis or other evidence of cord disease.

Crus and internal capsule.

Neuritis and hysteria.

Spinal vs Cerebral.

Traumatic neuroses and hysteria.

Hyperæsthesia and Hyperalgesia.—The well known hysterogenic zones of hysteria and neurasthenia furnish the best examples.* It may also be encountered in rickets, brain tumor, as a zone above the anæsthetic level in unilateral spinal hæmiplegia, or may be associated with meningitis, neuralgia and various toxic disturbances of the nerves.

Associated lesions.

Anæsthesia Dolorosa.—Is encountered in compression of the spinal cord in which it co-exists with analgesia but is associated with extreme pain. Analgesia and hyperalgesia may be encountered, the former suggesting hysteria and syringomyelia.

Cord compression.

TOPICAL DIAGNOSIS.

CORTEX.—Certain cerebral tracts are silent, i.e., symptomless, or may yield only psychic symptoms; *others permit direct localizing diagnosis.*

Monoplegia is the type and it may be limited to the head or an extremity, or in the case of the former, be confined to a single organ. On the other hand the *close proximity of motor areas may produce associated monoplegias* such as those of arm and leg, or face and arm or incomplete hemiplegias, *but usually with primary or ultimate predominate involvement of one area.* Complete *cortical* hemiplegia is possible, as a glance at the illustration will show, but is rare. *All unilateral cortical lesions produce paralysis of the side opposite the lesion.*

Incomplete hemiplegia common.

Opposite side affected.

Silent Areas.—The frontal lobe may be greatly damaged without symptoms as may the corpus callosum and corpus striatum, though in

* These are chiefly, the breast, ovaries (deep pressure), groin, spinal column and patella.

Few localizing signs in certain areas.

right handed persons *aphasia* may indicate a lesion of the left inferior frontal convolution, or *agraphia* one of the central convolution and it is probable that the *corpus striatum* is concerned in deglutition and contralateral muscle sense. A reference to the *motor areas*, fig. 210, will show the lesions that must follow irritation or destruction of any of them, i.e. *opposite facial paralysis, brachial monoplegia, crural monoplegia, hypoglossal paralysis*, etc., bilateral combined cortical lesions

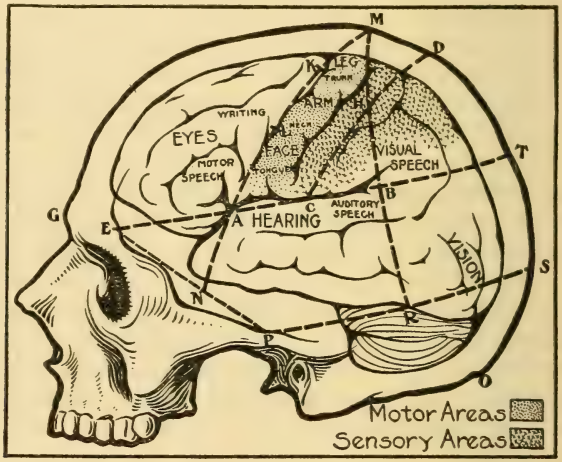


Fig. 210.—Motor and sensory areas of cortex. All lying anterior to the Rolandic fissure and above temporo-sphenoidal lobe are motor. The localization method of Chiene is also shown. This is applied as follows:—Find in the median line of the skull between the glabella (G) and the external occipital protuberance (O), the following points: The midpoint (M), the three-fourths point (T), and the seven-eighths point (S). Find also the external angular process (E) and the root of the zygoma (P) immediately above and in front of the external auditory meatus. Having found these five points, join EP, PS, and ET. Bisect EP and PS at N and R; also bisect AB at C and draw CD parallel to AM. The pentagon (ACBRPN) corresponds to the temporo-sphenoidal lobe, with the exception of its apex, which is a little in front of N. MDCA correspond to the Rolandic area, containing the fissure of Rolando, and the ascending frontal and the ascending parietal convolutions. A is over the anterior branch of the middle meningeal artery and the bifurcation of the Sylvian fissure; AC follows its horizontal limb. The lateral sinus at its highest point touches the line PS at R. MA corresponds to the precentral sulcus, and, if it be trisected at K and L, these points will correspond to the origins of the superior and inferior frontal sulci. The supramarginal convolution lies in the triangle HBC. The angular gyrus is at B. (Sherrington, Chiene and Grünbaum.)

Jacksonian epilepsy type.

being extremely rare. *Mere irritation* would produce the peculiar phenomena of Jacksonian seizures, viz.:—clonic convulsions in seat representing the motor point of maximum irritation and following in orderly succession the motor areas. This is usually unilateral though rarely bilateral through commissural transmission and is ordinarily

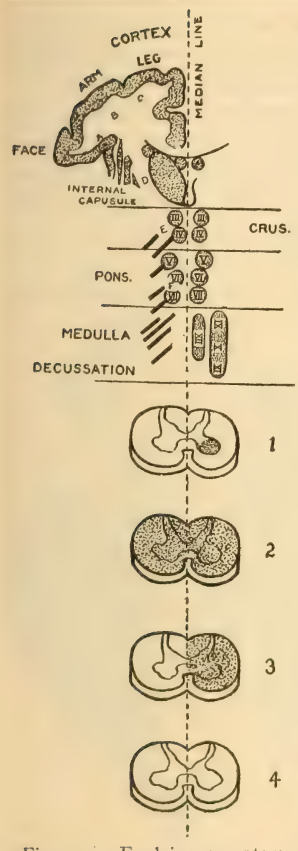


Fig. 211.—Explains symptoms caused by lesions affecting the motor tract in the brain and cord. Lesion at A, B, or C:—monoplegia of opposite side. Lesion at D:—hemiplegia of opposite side. Lesion at E:—oculo-motor paralysis of same side, hemiplegia of opposite side. Lesion at F:—facial and abducens paralysis of same side, hemiplegia of opposite side. Lesion of (1) anterior horns:—causes flaccid paralysis and lost knee jerks. Lesion at 2:—spastic paralysis of muscles below lesion if pyramidal tracts only are involved or the lesion is incomplete; flaccid paralysis if lesion is complete. Lesion at 3:—causes Brown-Sequard's paralysis. 4.—Normal cord. (After Van Gehuchten, modified.)

due to injury, rarely to paretic dementia. If the *parietal lobes* be involved there may be loss of sensation and of stereognostic sense on the opposite half of the body or ptosis (angular gyrus), conjugate deviation of the eyes or even visual aphasia and hemianopsia. *The occipital lobe* is so related to the optic tract as to produce hemianopsia, see p. 554, visual aphasia or even mind blindness. *The temporal lobe lesions* chiefly produce sensory aphasia, or, if bilateral, sound deafness.

Centrum Semiovale.—The structure and anatomical position of this great tract of projection and association fibres makes its lesions present either cortical or capsular symptoms and includes both marked sensory and the widest range of motor disturbances, including those of special sense.

Optic Thalamus.—It is said that the diagnosis of an isolated lesion of this ganglion depends chiefly upon the *loss of the facial expression* of *psychical emotion* upon the *opposite* side, the *voluntary* facial innervation being retained, a condition rarely observed. *Irritation symptoms* are sometimes present as in cortical lesions, as is crossed homonymous hemianopsia (pulvinar).

Lesions of the Internal Capsule.—(Fig. 212.) Almost all projection fibres between the cortex and the periphery (sensory, motor, and special sense) pass through this narrow and sharply defined area and any lesion entails serious consequences. *The anterior limb* is a symptomless area, but *the angular portion* (knee) is highly motor and *lesions therein*

cause paralysis in the opposite hypoglossal and lower facial distribution, if left sided, aphasia. If in the posterior ⅓ of the posterior limb *patchy hæmilateral anæsthesia,* and perhaps *hæmianopsia* and *auditory disturbances.* If in the anterior ⅔ of the posterior limb there is *hemiplegia of the opposite arm and leg. In most instances the paralysis is extensive and results in a combination of these lesions,* i.e., complete hæmiplegia. The upper branches of the facial, the trunk muscles, those of the neck, eye, and of mastication are paralyzed only in bilateral lesions.

The Crura.—At this level the mixed type of paralysis commences and extends throughout the pons and medulla, the relation of the pyramidal tract to the cranial nerve nuclei being such that the lesion may produce spastic paralysis of the lower segment (brachio-crural type) and a flaccid paralysis of cranial nerve areas. Thus, lesions of the crus are associated with hemiplegia with *crossed* oculomotor paralysis, sometimes including partial paralysis of the 3rd and 4th nerves of *the other side* through basal exudate. The type of *tegmental* lesion is hæmiataxia with crossed oculo-motor paralysis (Osler).

Gendrin's type.

Hemiplegia.

Involves 3d nerve.

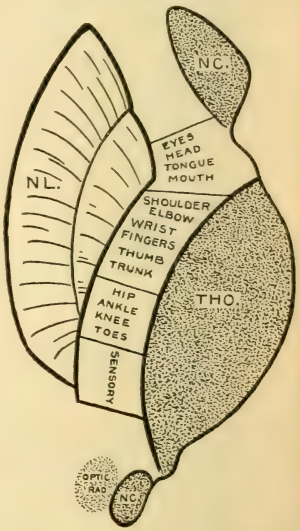


Fig. 212.—Topography of motor tract in internal capsule.

Lesions of the corpora quadrigemina are characterized by *flaccid (nuclear or tract) oculo-motor paralysis, 3d and 4th nerves, stumbling gait and ataxia* (cerebellar type).*

Hemiplegia.

Varies with site.

Lesions of the Lower Pons.—The ordinary type of paralysis is crossed spastic hemiplegia, with paralysis of facial and trigeminus on side of lesion (muscles of mastication, anæsthesia of trigeminal distribution, facial paralysis). *Abducens paralysis (external rectus same side, and internal opposite lesion),* defective taste and articulation (facial and

* The region is rich in nuclei and lesions may be unilateral or bilateral and involve the tegmentum (incomplete hemianæsthesia), the optic tract or lateral geniculate body (hemianopsia), the crusta (paralysis of opposite leg and arm or even hypoglossal and facial hemiplegia) or produce defective hearing through involvement of the median geniculate body (Jakob).

hypoglossal fibres), brachio-crural hemiplegia of opposite side and perhaps ataxia, vertigo, or trismus are also encountered.

Lesions of the Medulla Oblongata.—Here again lesions may be unilateral or bilateral. The *type* is:—*brachio-crural hæmiplegia* and *hæmianæsthesia* of the opposite side with tongue *pointing towards lesion*, or if the fillet be involved bilateral anæsthesia. Anarthyria or dysphagia results and the type of *glosso-labio laryngeal paralysis* may be present and is usually bilateral. Coarse lesions rapidly fatal.

Coarse lesion.

Involves lower cranial nerves.

Lesions of the Cerebellum.—Though sometimes lacking symptoms if unilateral, the chief and commoner characteristics of involvement

May be symptomless.

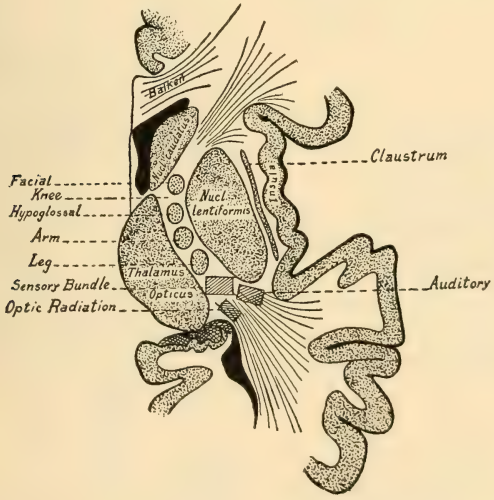


Fig. 213.—Showing relative position of motor and sensory fibres in the internal capsule. (After Monakow.)

of the *vermiform process* are cerebellar *ataxia* (staggering, drunken gait), vomiting, headache, *vertigo* or even movements of forced body rotation. Nystagmus and optic neuritis are common. Inco-ordination is coarse and *may be greatly diminished in recumbency with closed eyes.*

Ataxia and vertigo.

SEGMENTAL PARALYSES.*—The paralysis of muscle groups is the basis of localization in diseases of the cord and peripheral nerve lesion, and the diagrams (209, 216) show the lack of correspondence between the point of nerve emergence and its segmental origin and the seg-

*Starr, Mills, Sachs, Dana, Thorburn, Butler and Osler.

mental sensory areas. So far as possible this topic will be dealt with on the basis of segment *symptomatology* preceded by a discussion of lesions affecting certain of the cranial nerves.

The Tongue.—(Hypoglossal nerve). In bulbar palsies, and basal processes deviation of the tongue to the paralyzed side (genioglossus), loss of backward movement (styloglossus) or deviation towards the sound side when retracted, or paralyzed side when protruded, associated perhaps with atrophy, unilateral or bilateral (lingualis), may be encountered.

The Uvula and Velum Palati.—(Pharyngeal plexus, possibly 7th nerve). Nasal voice and regurgitation of food through the nose indicates

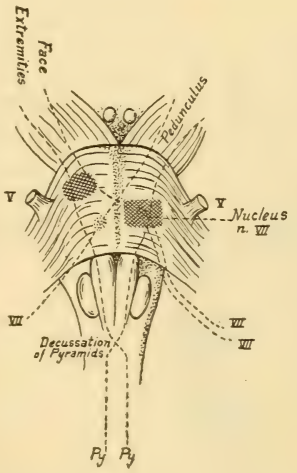


Fig. 214.—Illustrating the mechanism of crossed paralysis in pontine lesions. (After Hermann.)

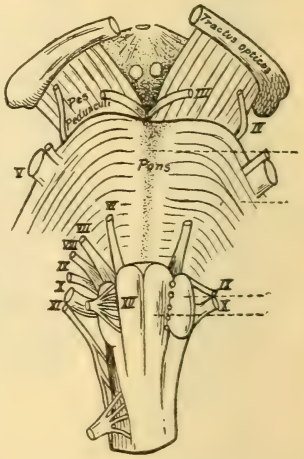


Fig. 215.—Ventral aspect of medulla oblongata. (After Edinger and Hermann modified.)

bilateral palsy; deflection towards the sound side, unilateral palsy of the azygos uvulæ. Food regurgitation and immobility in "ah" intonation (paralysis involving the levator palati). Food regurgitation and nasal speech are also seen in palato-pharyngeus paralysis (5th nerve).

Pharynx and Larynx.—Choking in deglutition due to the entrance of food into the larynx may arise from the failure of epiglottis closure due to paralysis of the stylo-pharyngeus (glosso-pharyngeal nerve), or deficient downward movement due to paralysis of the constrictors (pharyngeal plexus).

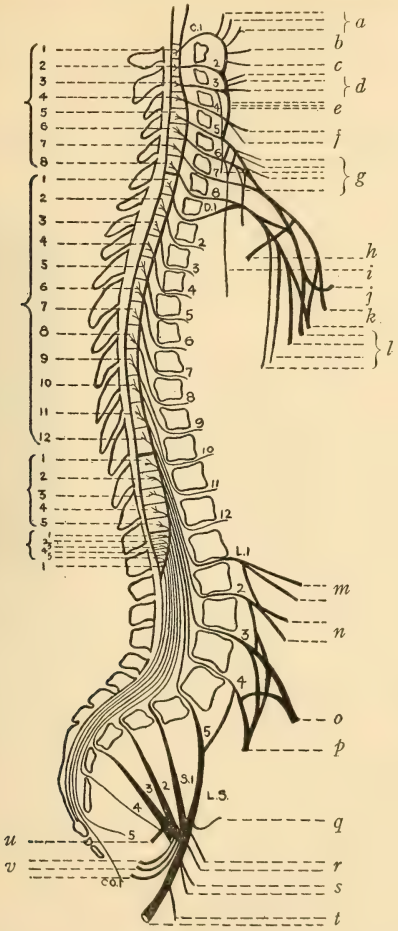


Fig. 216.—Segments of spinal cord and their relation to the vertebral landmarks. The variation between the level of origin of the spinal nerves and their point of exit is well shown. The numerals indicate the segments, those to the right the spinal nerves, and corresponding vertebræ. The lettered nerves when grouped will be named in their order a, nerves to rectus lateralis, to rectus anticus minor, anastomosis with hypoglossal; b, anastomosis with pneumogastric; c, nerve to rectus anticus major; d, to mastoid and great auricular and transverse cervical; e, to trapezius, ang. scap. and rhomboid; f, supra-clavicular and supra-acromial; g, phrenic, lev. ang. scap. to rhomboid sub-scapular and sub-clavicular; h, to pectoralis major; i, posterior thoracic, serratus magnus; j, circumflex; k, musculo-cutaneous; l, median, radial, ulnar internal cutaneous and lesser internal cutaneous; m, ilio-hypogastric and ilio-inguinal; n, external cutaneous and genito-crural; o, anterior crural; p, obturator; q, superior gluteal; r, to pyriformis and gemellus superior; s, to gemellus inferior and quadratus; t, lesser and great sciatic; u, to levator ani; v, to obturator int., to sphincter ani and coccyx. (Dejerine and Thomas.)

Hoarseness, aphonia or paralytic stenosis and the various faulty positions of the cords as shown in the illustration may be due to paralysis of the musculature (recurrent laryngeal nerve excepting for the crico-thyroid).

Spasm or paralysis of the sterno-cleido-mastoid (*spinal accessory, medulla, 1st, 2nd, 3rd, cervical segments*) may cause wry neck (spasm), the head being inclined to one side with chin raised and pointing to the opposite shoulder. If paralyzed, face cannot be turned to opposite side or if bilateral the head cannot be raised from the bed in recumbency.

Fig. 217.—Vocal cords. (Diagrammatic mirror picture.)
1. Normal position in breathing and phonation respectively.
2. Adductor paralysis (left) 2′. Bilateral adductor paralysis. Both in phonation.
3. Unilateral abductor (left) and 3′ bilateral abductor paralysis both during breathing.
4. Left recurrent paralysis phonation, 4′ same in respiration, 4″ recurrent bilateral in both respiration and phonation.
5. Arytenoid paralysis phonation. 5′ Thyro-arytenoid paralysis, phonation 5″ Arytenoid and thyro-arytenoid paralysis.

Flexion, Rotation of the Head.—If the chin cannot be brought to the chest the rectus capitis anticus, major and minor are at fault. Failure of rotation can seldom be traced to the rectus capitis lateralis but is usually due to sterno-mastoid paralysis.

Deficient Thoracic Respiratory Movement affecting elevation and lateral movements of the ribs suggests paralysis of the scaleni (*lower cervical segments*).

Deficient Upper Spine Flexion.—Suggests lesions involving the longus colli (lower cervicals). Impaired power to raise chin may be due in part to a lesion involving the clavicular portion of the trapezius (spinal accessory); a depressed shoulder to a lesion of the middle portion; abduction of the scapula to a lesion of the lower portion (*spinal accessory, 2nd and 3rd cervical segments*).

Loss of power to approximate scapulæ indicates a lesion of the rhomboids (*4th and 5th cervical segments*).

Oblique Position of the Scapula.—The inferior angle approaches the median line, the bone is raised, the arm cannot be raised above the horizontal position and if stretched forward the scapula projects (alar scapula). This indicates paralysis of the serratus magnus (*posterior thoracic nerve, 5th and 6th cervical segments*).

Inability to Raise Arm though Shoulder Rises.—The latter is flattened from atrophy and a groove appears below the acromiom.

This indicates paralysis of the deltoid, any one of the three divisions of which may be separately affected (*circumflex nerve, 4th, 5th and 6th cervical segments*). If the arm cannot be moved outward, the infraspinatus is involved (suprascapular). If writing is difficult the teres minor is involved (circumflex), if the arm cannot be moved inward a lesion of the subscapularis is suggested (*subscapular nerve*). All three represent the 4th, 5th and 6th cervical segments.

Impaired Lateral Trunk Movement.—With failure of dorsal spine extension and backward movement of the arm indicates a lesion involving the latissimus dorsi (*subscapular and 6th, 7th cervical segments, chiefly*).

Inability to Firmly Proximate the Volar Surfaces with Extended Arms.—Suggests a lesion of the pectoralis major (*anterior thoracic nerve, 5th, 6th and 7th cervical segments*).

Inability to extend arm against resistance indicates a lesion of the triceps which is associated with liability to subluxation of the humerus, spontaneous or induced by slight causes (*musculo-spiral, 6th, 7th and 8th cervical segments*).

Impaired flexion of the arm suggests a lesion of the biceps (*musculo-cutaneous, 4th, 5th and 6th cervical segments*), if flexion and pronation are both deficient the supinator longus is to be considered. The arm is usually spindle shaped from atrophy of the muscle (*musculo-spiral, 4th, 5th and 6th cervical segments*).

Deficient supination with arm extended indicates supinator brevis involvement (*musculo-spiral, 5th cervical segment*).

Failure of dorsal flexion and abduction of the wrist with atrophy of the forearm indicates a lesion of the extensor carpi radialis longus and brevis; if adduction also fail from paralysis of the extensor carpi ulnaris drop wrist is produced; if extension and abduction of the first phalanges fail a paralysis of the three extensors of the fingers is indicated (*for all musculo-spiral, 7th cervical segment*).

Impaired flexion of the fingers and wrist suggests paralysis of the flexors of the wrist and fingers. The median nerve supplies the flexor carpi radialis, palmaris longus, flexor sublimus digitorum, and, in part, the deep flexor of the digits, the ulnar supplying the remainder of the last named and the flexor carpi ulnaris, all being related to the *8th cervical segment.*

Failure of Abduction and Adduction of Fingers.—Suggests a lesion of the interossei and lumbricales (*ulnar and median, 8th cervical and 1st dorsal segments*). With extension of the first phalanges and

flexion of the 2nd and 3rd and deep interosseus spaces it forms the claw hand (*main en griffe*).

Atrophy of the Ball of the Thumb.—An impairment of its extension and adduction suggests a lesion of the extensor pollicis brevis and thenar muscles to which is added flexion of the second toward the first phalanx if the extensor pollicis longus is involved, with deficient abduction if the abductor pollicis longus is affected (for all, *musculo-spiral nerve, first dorsal segment*). If flexion of the thumb fails and atrophy is present the "*ape hand*" is formed (abductor brevis pollicis, flexor brevis and adductor pollicis). The opposing movement may be absent (opponens pollicis and in part flexor brevis and abductor pollicis brevis) or flexion of the terminal phalanx may fail from paralysis of the flexor pollicis longus. The nerve supply is from the *median and ulnar and first dorsal segment.*

Lower spinal lordosis with deflexion towards sound side in unilateral lesions indicates a lesion involving the erector spinæ, sacro-lumbalis and longissimus dorsi (*dorsal nerves, 2nd to 12th dorsal segments*).

Lordosis with Prominent Nates and Abdomen.—Inability to rise up from a dorsal recumbent position without assistance from hands indicates a lesion of the abdominal muscle group (*dorsal nerves, 2nd to 12th dorsal segments*).

Imperfect lateral movement of lower spine suggests quadratus lumborum involvement (*lumbar nerves and segments*).

Loss of Thigh Adduction.—(Thigh rolls outward), indicates paralysis of the adductors (*obturator nerve, great sciatic and crural, 3rd lumbar segment*).

Imperfect flexion of the thigh suggests involvement of the sartorius (*crural nerve, 3rd lumbar segment*).

Impaired Leg Extension.—Involvement of the quadriceps femoris (*crural, 3rd lumbar segment*).

Impaired flexion and difficulty in rising from the horizontal position suggests involvement of the ilio-psoas (*crural nerve, 4th lumbar segment*) and the tensor fasciæ femoris (*superior gluteal, 4th lumbar segment*).

Impaired Outward Rotation.—Leg being turned inward suggests involvement of the external rotators, pyriformis, gemelli and quadratus femoris supplied by *sacral plexus, 5th lumbar segment*, as well as the internal and external obturators (*obturator nerve, lumbar plexus*).

Complete loss of thigh extension and abduction with waddling gait and inability to climb indicates paralysis of the gluteal muscles

(*inferior gluteal nerve, sacral plexus, first and second sacral segment and superior gluteal, 1st and 2nd sacral segments*).

Impaired flexion perhaps associated with hyperextension through action of quadriceps and hyperflexion in standing, points to a lesion affecting the biceps, semitendinosus and semimembranosus.

Deficient foot flexion, i.e. patient cannot stand on tiptoes or raise heel indicates gastrocnemius, plantaris and soleus paralysis (*internal popliteal nerve, 5th lumbar segment*).

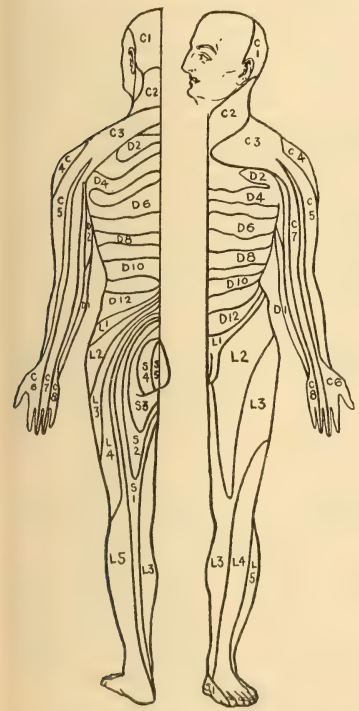


Fig. 218.—Areas of anæsthesia following segmental lesions of spinal cord, the numbers shown corresponding to the affected segment. (After Starr.)

Drop Foot.—Usually with excessive knee and hip flexion with perhaps pes equinus or equinovarus from contracture points to involvement of the anterior tibial muscles, i.e. tibialis anticus, extensor digitorum and extensor longus pollicis (*anterior tibial nerve, 5th lumbar and 1st sacral*).

Deficient Abduction.—Tendency to flat foot increased by contracture points to involvement of the peroneus longus (*peroneal nerve, 1st and 2nd sacrals*). Deficient abduction with resulting deformities suggests involvement of the tibialis posticus *posterior tibial nerve, 1st and 2nd sacrals*), and the peroneus brevis (*peroneal nerve, 1st and 2nd sacral segments*).

Adduction of Toes.—Paralysis of interossei with hyperextension of first phalanges and flexion of the 2nd and 3rd (claw foot) indicate involvement of the interossei and lumbricales (*posterior tibial, 1st and 2nd sacral segments*).

Deficient Toe Flexion.—Impaired power to push foot off ground indicates a lesion of the adductors, flexor brevis and abductor hallucis.

The segmental sensory localization is indicated by fig. 218.

BRACHIAL PLEXUS PARALYSIS.—Paralysis is usually the re-

sult of traumatism or compression of some sort most commonly below the clavicle, and may be either partial or complete. If only the upper portion be involved the lower muscle groups (forearm and hand) may with the exception of the supinator longus escape (Erb's type). Complete lesions involve a total motor and sensory paralysis of the arm and there is a lower arm type in which forearm extension and use of the hand are lost, the triceps, flexors of the wrist, pronators, extensors and flexors of the fingers being involved and frequently ocular signs of involvement of the sympathetic.

Erb's type.

Klumpke's type.

Fig. 219.—Drop wrist.

CERTAIN SPINAL NERVES.—Occipital.—Neuralgia, anæsthesia.

Posterior Thoracic.—Arm cannot be raised above horizontal and rotated. Projecting scapula (serratus magnus).

Anterior Thoracic.—Inability to adduct arm (pectoralis major).

Musculo-cutaneous.—Loss of elbow flexion power (biceps, brachialis anticus). Anæsthesia inner border of forearm.

Circumflex.—Loss of outward rotation and elevation of arm (deltoid teres minor), third head of triceps.

Suprascapular.—Impaired outward shoulder rotation and elevation.

Musculo-spiral and Radial.—Paralysis from lead, arsenic or alcohol, tumor and crutch pressure and traumatism or whatever cause is common and leads to the characteristic dropping of the wrist from extensor paralysis (drop wrist) so commonly seen in clinics. A factor in differential diagnosis is the involvement or non-involvement of the supinator longus which is usually spared in lead poisoning. The triceps and anconeus are paralyzed, extension of forearm lost and there is wasting of the back of the arm.

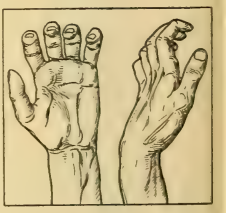


Fig. 220.—Claw hand.

Median.—Abduction of thumb and flexion of thumb and first and second fingers lost. Thenar and anterior forearm atrophy usually marked. If forearm be flexed pronation is impossible. The thumb and index finger are approximated and there is paralysis of the abductor pollicis. It is seldom paralyzed alone.

Ulnar Nerve.—Impaired abduction and flexion of hand (flexor carpi ulnaris and part of flexor sublinis digitorum) wasting of ball of little finger and interosseous spaces, one cannot cup the palm, nor span. The ring and little fingers are "clawed" ("claw hand," "main en griffe"). Paralysis of their lumbricales and interossei prevent flexion of the proximal or extension of the distal phalanges and the unopposed muscles exaggerate the position.

Fig. 221. Dropped foot.

Intercostal Nerves.—Violent neuralgic pains or other symptoms of irritation, sometimes herpes zoster along the course of affected nerve, anæsthesia.

Crural Nerve.—Patient cannot stand or walk and tendon reflex is absent (psoas-quadriceps extensor).

Obturator.—The obturator plexus is a frequent seat of pain, referred to the inner side of the thigh and knee joint in disease of the mid-lumbar, sacro-iliac and hip joints. Obturator paralysis means loss of adduction.

Sciatic.—Paralysis of leg flexors, foot and toe, sciatica.

Peroneal Nerve.—Foot drop from paralysis of flexors of foot or leg by contracture, talipes equinus or varus may appear.

Tibial Nerve.—Paralysis of calf muscles, loss of foot extension or toe flexion. Talipes if contractures occur.

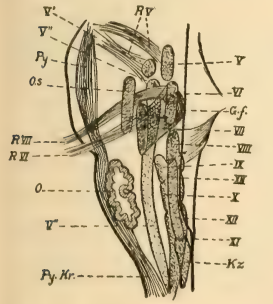


Fig. 222.—Lateral aspect cranial nerve nuclei within medulla oblongata. (After Erb.)

THE CRANIAL NERVES.—*Irritation produces excess or perversion of functional activity*, i.e. spasm, tremor, pain, itching, formication, etc., *destructive lesions*, ultimate loss of function, muscle atrophy, etc., inasmuch as a *peripheral* neurone is thus disturbed or destroyed. The nucleus of the cranial nerve corresponds to the nutrient anterior horn cell of the spinal nerve.

Irritation vs. Destruction.

OLFACTORY NERVE.—Test.—Apply to each nostril in turn bottles containing well known aromatic oils (peppermint, cloves, assafetida, bay, etc.) and have the patient describe the odor. *Any inflammation, degeneration, necrosis, traumatism or pressure affecting the terminal filaments, bulb or tract from the uncinate gyrus and thalamus to the Schneiderian membrane produces lessened, absent (anosmia) or perverted (parosmia) function.* These constitute also symptoms of hysteria, epilepsy, locomotor ataxi-

Simple tests.

Anosmia. Parosmia.

Hallucinations.

and insanity (hallucinations of smell). *Trigeminal paralysis and certain catarrhs* act no doubt by lessening the secretion.

THE EYE, ITS REFLEXES AND THE OPTIC NERVE.—The Pupil.—*The shape, equality of the two sides and the mobility or response to light and accommodation should be noted. Irregularities in outline* suggest chiefly adhesions from past iritis. In *acute iritis* the color of the iris is turbid and greenish, the corneal circumference showing an hyperæmic zone. Difference in the color of the iris of the one eye as compared with the other is especially important in detecting iritis in eyes normally dark. The commoner causes of iritis are gout, rheumatism, trauma and syphilis and the first *two* are usually of the unilateral relapsing type, the last is bilateral, belongs to the period of secondary infection and seldom recurs.

Past iritis.

Acute iritis.

Syphilitic iritis.

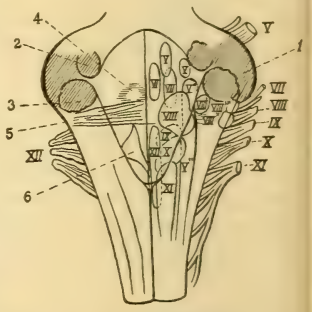


Fig. 223.—Nuclei of cranial nerves in medulla oblongata. (After Erb.) The nuclei by Roman numerals. The nerve roots are similarly shown at the side.

1. Brachium pontis.
2. Brachium conjunctivæ.
3. Cerebellar peduncle.
4. Eminentia teres.
5. Striæ acousticæ.
6. Ala cinerea.

Inequality.—Even in healthy persons, considerable inequality occurs but *pathologic* irregularity is important and may represent either unilateral contraction or dilatation. The former (*myosis*) may be due to *iritis, oculomotor irritation or paralysis of the sympathetic, but occurs normally with suspended reflexes, e.g. in sleep.* The latter (*mydriasis*) indicates conversely *sympathetic irritation* (aneurism of the arch, goitre, enlarged glands or other swelling, etc.) *or oculo-motor paralysis.* Furthermore, glaucoma, cataract, disease of the optic nerve and the retinal hæmorrhage of albuminuria may be the causes of dilated pupil, both unilateral and bilateral.

Oculomotor and sympathetic.

Local disease.

Persistent mydriasis is frequently noted in cases of hysteria, high myopia, neurasthenia, severe dyspnœa or actual asphyxia, cerebral abscess, hemorrhage, thrombosis and tumor, exophthalmic goitre, epileptic coma, catalepsy, melancholia, mania and active delirium, late meningitis, in aortic regurgitation, shock, fear or strong emotion generally, nausea, and in poisoning by such mydriatics as tobacco, alcohol, nitrous oxide gas, cocaine, choral, chloroform, ether, duboisin, hyoscin, scopolamin, stramonium, conium and belladonna. Certain cases of locomotor ataxia show as an early symptom dilated pupils and

Early eye changes in tabes.

ptosis with beginning atrophy of the nerve head in many instances and *such cases run a course marked by long delay in the development of ataxic and paralytic symptoms.*

Contracted Pupils.—The following conditions may be associated with persistent bilateral miosis. Locomotor ataxia, general paresis, high hypermetropia or astigmatism, disseminated sclerosis, tumor, hemorrhage, meningitis, inflammation or degeneration of the brain or cord, uræmia, sunstroke (early stage), photophobia, congestion of the iris, etc.

The primary action of ether, chloral, opium, eserine and pilocarpine is miotic and general venous congestion, unless associated with marked dyspnœa, may produce bilateral pupillary *contraction.*

Unilateral Contraction.—Marked *unilateral* contraction aside from adhesions, iritis, paretic dementia and apoplexy is an important suggestive sign of a serious lesion of the brain, cervical cord or any region involving the motor oculi or sympathetic. Mediastinal tumors, aneurismal, glandular or malignant, with their pressure effects, account for many cases, and should be constantly borne in mind. In not a few cases an artificial eye has puzzled the careless physician. Tuberculous cases often present persistent slight dilatation, usually unequal and sometimes merely transient.

Often a serious symptom.

Trivial.

Hippus.—An oscillating contraction and dilatation of the iris on sudden exposure to light is of little clinical importance, though often associated with nystagmus and sometimes present in disseminated sclerosis.

EYE REFLEXES.—Light response: *Proper testing demands that the consensual light reflex be remembered and involves both light and accommodation reactions*, that of convergence being of no special importance. The best methods demand the use of the ophthalmoscope in a darkened room and the use of such a + lens as will enable the observer to see the magnified pupil as the mirror flashes the light upon the eyes. Each must be separately tested and the patient should look slightly to the right or left of the observer to avoid the accommodation reaction. As a matter of fact there is normally a sympathetic reaction of the other pupil (consensual reaction).

Direct and indirect.

Reaction to Accommodation.*—*Ordinarily contraction of the pupil attends accommodation for near vision and in locomotor ataxia and general paralysis especially, this reflex persists* though that for light is lost (*Argyll Robertson pupil*). In general such a pupil indicates

Argyll Robertson pupil.

* Probably inseparable from the convergence reaction.

a lesion of the optic tract or Meynert's fibres, whereas *fixed contraction* indicates a lesion of the centre or motor oculi.

OPTIC NERVE.—*The tiny primary optic neurones** receive the visual impressions through their dendrites, the retinal rods and cones, *the axones* passing to the *visual centres* in the optic thalamus (pulvinar), corpora quadrigimini and geniculate bodies, through the posterior portions of the internal capsule and finally, in the optic radiation, to the cuneus. *The axones from the nasal sides of the retina, which represent the outer and larger portion of the visual fields, cross and pass to the centres of the opposite side. Those of the temporal portion (inner and lesser visual field) run direct to the centres of their own side. The point of decussation* (chiasma) *therefore represents all fibres.*

Course of fibres.

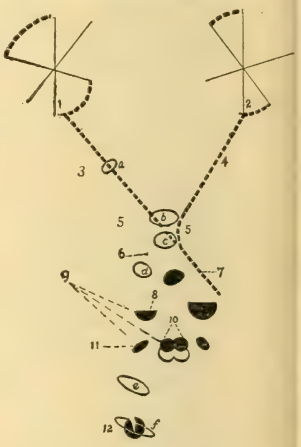


Fig. 224—Course of optic nerve fibres from cortex to retina showing relation of visual fields. (After Sahli, slightly modified.)

Explanation. A lesion at *a* produces unilateral blindness, at *b* or *c* a temporal hemiopia by interruption of the nasal fibres, at *d*, *e* or *f* a lesion will produce right homonymous hemiopia. *1* and *2* represent the retina; *3* and *4*, optic nerves; *5*, the chiasm; *6* and *7*, optic tracts; *8*, the pulvinar; *9*, the primary optic centre; *10*, the anterior corpora quadrigemini; *11*, the external geniculate body; *12*, the occipital lobe. The right and left tracts, their respective retinal distribution and visual fields are indicated by the solid red (left tract) and dotted black (right tract) lines.

Visual fields.

Anterior to it lie the mixed fibres for the respective halves of each eye; posteriorly, those for the outer segment of the eye of the same side (nasal visual field) and the inner portion of the opposite eye (temporal visual field).

Hemiopia.

Interference with these fields *causes hemianopsia* and the *variety of the hemianopsia* and the *naming of the affected visual field* represent the *patient's view point;* hence *the variety of hemiopia is the reverse of the retinal field affected,* i.e. *temporal retinal change =nasal hemianopsia,* etc.† *The condition may be unilateral or bilateral, total, partial or concentric,* affecting *corresponding halves* (homonymous), *both temporal or both nasal fields* (heteronymous), or even *both upper or lower halves* (superior or inferior altitudinal).

Effect of lesions.

A lesion may occur at any point from and including the retinal surface to the cortical centres. If unilateral and in front of the *chiasm* blindness

* The true optic nerve (Church and Peterson).

† Hemiopia, hemianopsia, hemianopia are synonymous terms.

of that eye follows. If *at the chiasm* either the anterior or posterior angle (nasal ½ of retina) is usually affected causing bitemporal hæmianopsia. If *the outer portion* only be involved unilateral or bilateral nasal hemianopsia follows. A lesion behind the chiasm if on the right side causes left hemianopsia, if on the left right hemianopsia.* Precisely the same phenomena occur if a lesion affects the more central tracts, save that the difficult "hemianopic" (Wernicke's) pupillary reaction is to be considered. *Test.* Carefully note size of pupils by plane mirror illumination in dark room, then with the ophthalmoscope direct a narrow, strong beam of light upon the *blind portion* of the retina. If *pupillary contraction* occurs the lesion is posterior to the corpora quadrigemina.† It should be remembered that (a) *tract lesions ordinarily produce hemianopsia,* (b) *nerve lesions total blindness* and that (c) *central vision is retained in unilateral tract lesions* because *the macula of each eye receives fibres from both optic tracts.*

Wernicke's localizing reaction.

General laws of localization.

Diagnostic Significance.—Disturbances of vision *due to tract lesions* are usually associated with *gummata, new growths and syphilitic and tuberculous meningitis.*

Causes of tract lesions.

Associated Lesions.—Incomplete hemianopsia with aphasia and mind or word blindness indicates a *cortical lesion. Angular gyrus lesions* are suggested by impaired vision in one eye associated with a contracted visual field of the opposite eye. *Athetosis* suggests a *pulvinar lesion; altitudinal hemianopsia, a lesion of the upper or lower part of the chiasma,* or if unilateral, a *lesion of the cuneus. Hysterical hemianopsia is associated with the usual stigmata, hemianæsthesia, insensitive conjunctiva,* and *more often takes the form of contracted fields and alteration of color vision areas.* Fugitive hemianopsia may accompany *migraine. Total unilateral blindness may be due to a destructive lesion of the occipital lobe or optic nerve. Bilateral lesions of the cuneus or optic nerves or total destruction of the chiasma will cause bilateral blindness. High refractive errors* of long standing may be associated with unilateral loss of vision.

Cortical lesions.

Angular gyrus.

Pulvinar.

Chiasma and cuneus.

Hysteria.

Total blindness.

Color Fields.—The limits of color vision may be indicated by concentric circles, the inner and least being for green, then red, blue and finally white. Aside from actual nerve lesions color perception is greatly modified in toxic amblyopias (tobacco, etc.) and the neuroses.

Vary with color.

Modifying conditions.

* For example, a lesion of the right tract cuts off the retinal impressions of its own temporal ½ and the opposite nasal half of the retina, hence left visual fields are lost for both eyes (left homonymous hemianopsia).

† As the pupillary reflex sensory fibres run in the tract nearly to the corpora any lesion anterior means an interrupted reflex arc.

Amblyopia, Amaurosis.—*Amblyopia and amaurosis* describe respectively, the one, impairment, the other, total loss of vision without actual lesions, but in the latter associated with various neuroses and toxæmias. *Amaurosis* is alarming but usually *transitory*, lasting from a few hours to several days. It suggests uræmia, diabetes, severe and especially *acute* anæmias, spasmodic contraction of retinal vessels, hysteria and migraine, and has followed the administration of quinine and the salicylates, the over-use of alcohol and tobacco, cerebral trauma and lead poisoning.*

Transitory blindness.

Total blindness.

Testing Vision.—Acuity.—If total blindness be suspected the patient should be asked to count the fingers of the physician as held before him at varying distances, failing in this he should be tested for light perception in a dark room.† (See also simulated blindness, p. 624.)

The Visual Field.—Rough Test.—Squarely face the patient at a distance of eighteen inches. If the left eye is to be tested the right should be covered and the patient told to look squarely and fixedly at the right eye of the examiner whose left is closed. The latter then holds his open hand well off to the side but on a level with the eye and brings it inward, moving the fingers constantly until the patient sees them *without moving the head or eyeballs;* the same procedure tests all portions of the field and checks the patient's range with the presumptively normal one of the physician. For more accurate or graphic delineation the various perimeters may be employed.

Lacking special instruments.

Central Scotomata.—*Loss of central vision or central amblyopia if unilateral is usually due to choroiditis or retinitis, if bilateral to chronic toxæmias and syphilis.* Scotomata of color vision are best detected by the perimeter.‡‡ In migraine and certain conditions of cerebral meningeal irritation the so called "flittering" scotomata appear. These are "like a sunset cloud." *Muscæ Volitantes.* Little floating motes, specks or thread-like figures are common in over-strain, hysteria, anæmia, dyspepsia, eye-strain and various other conditions.

Unilateral vs. Bilateral

Scotomata.

Muscæ volitantes.

* *Unilateral* amblyopia suggests either "amblyopia ex-anopsia" of strabismus or a preexisting deviation which has become straightened, or the lesions that may produce complete blindness, or affecting the angular and supra-marginal convolutions.

† Minor disturbances are tested by the use of the well known Snellen's test types.

‡ A rough test consists in holding before the patient's eyes at a distance of 1½ to 2 feet a black square of cardboard with a central white spot. While his eyes are fixed on the white spot a black strip carrying a green or red spot or wafer at its extremity is placed quickly at the outer side of the white. If not seen it indicates central color scotoma.

PLATE V

Fig. 1. Normal Fundus

Fig. 2. Embolism of the Central Artery

Fig. 3. Thrombosis of the Central Vein (So-called Hemorrhagic Retinitis)

Fig. 4. Albuminuric Retinitis

(From Thorington's "Ophthalmoscope and How to Use It")

DESCRIPTION OF PLATE V.

FIG. 1.—NORMAL FUNDUS OF LEFT EYE.

Direct Method. Disc is round, having light-colored centre, distinct margins and yellowish-red intermediate zone. Choroidal ring almost complete and slightly more pigmented to the temporal side. Two cilio-retinal vessels on the lower outer edge of disc passing toward the macula. The crescentic fovea centralis, with surrounding blood-red area, is unusually well shown and most typical at this young age. Veins and arteries are slightly to the nasal side of the disc. The arteries cross the veins on the disc, but in the periphery the veins cross the arteries.

FIG. 2.—EMBOLISM OF THE CENTRAL ARTERY OF THE LEFT EYE. DIRECT METHOD.

Large, oval-shaped, blanched area, which includes the disc and macula. Cherry-red spot at the macula. Intermediate zone of the disc shows apparently normal except for a faint fogginess. The arteries are almost empty and the smaller ones have the blood stream broken in different places. The arteries have lost their light streak. The veins are slightly distended, but not tortuous; the light streak is almost lost in the veins, but can be faintly seen.

FIG. 3.—THROMBOSIS OF THE CENTRAL VEIN OF THF LEFT EYE. (APOPLEXY OF THE RETINA, HEMORRHAGIC RETINITIS.)

Fundus Changes. Left Eye. Acute papillitis. Disc very much swollen and apex seen with +4 D. and fundus without any lens at the sight hole. Arteries small and very few of them in view. The veins are very tortuous, looking like half hoops or serpentine, hence the condition as described in the text of "Medusa Nerve." The light streak in the veins is conspicuous at the top of each loop. The hemorrhages are of all sizes and shapes and shades of red.

FIG. 4.—ALBUMINURIC RETINITIS. BRIGHT'S DISEASE. RIGHT EYE. DIRECT METHOD.

Fundus Changes. Swollen disc, striated edges; exudation ("snow banks") about its edges with two areas above and one below the disc. Macular figure unusually well marked. Many scattered and flame-shaped hemorrhages seen in the periphery aud about the disc. Vessels about the disc show effusion into their sheaths by the white edge at each side of the vessel. The disc resembles that of choked disc in brain tumor, but the "snow banks" and macular figure are almost too conspicuous for such a diagnosis.

Optic Neuritis.—(Choked disc, papillitis). Amongst the varied lesions shown by the accompanying plates optic neuritis easily holds first place. It occurs in from 80–90% in all brain tumors, is usually found in meningitis, especially if that be basal and is not infrequently present in cerebral abscess. In all cerebral lesions it is likely to be bilateral, though more advanced in one eye than in the other. If in such cases it is unilateral it is on the side of the brain affected. As regards the location of brain tumors in relation to the frequency of optic neuritis, it may be said that it is invariable in tumors involving the corpora quadrigemina, occurs in 90% of those at the base, parieto-occipital region and cerebellum, and with scarcely less frequency in those of the crura and frontal lobes. In the case of no region does it fail to appear in much less than 50% of the lesions. Amongst other causes are chronic nephritis, diabetes, hydrocephalus, sinus thrombosis, syphilis, lead poisoning, cysts, meningeal hemorrhage and trauma; rarely it is noticed in severe acute infections and sunstroke. Its characteristics are so well shown by the plates as to need no further description. (See also p. 579.)

Important.

Unilateral vs. Bilateral.

Brain tumor.

Other associated lesions.

OPHTHALMOSCOPY.—Every physician should be able to use the ophthalmoscope at least so far as its findings relate to the common lesions aside from actual refraction errors. It often proves the master key of diagnosis in obscure cerebral lesions, syphilis, tuberculosis, locomotor ataxia and arterio-sclerosis, and of prognostic value in diabetes and chronic nephritis. Any good form of ophthalmoscope will suffice, the difference being rather in the skill and cerebration of the physician than in any marked advantages possessed by one over another instrument. Not only is the instrument indispensable for retinoscopy and refraction but hardly less so for the examination of the medial and external structures.

Often the master key.

Oblique Illumination.—A beam of light is focused obliquely upon the corneal surface by an indirect examination lens, which is held 2 to 4 inches above and to the side of the affected eye, slightly in front of the patient; a second lens may be used for magnification. *Opacities of the cornea* appear as small cloudy or opaque areas (*nebulæ*) or larger ones (*leucomata*). *Deep seated opacities* such as occur most often in syphilis *yield a reflection, superficial areas are dull.* The presence or absence of the *light reflex* should be noted and the *consensual reflex* tested. *Hippus, photophobia* and the *pupillary contraction of accommodation and convergence,* are readily tested and the *pupillary outline* and its *bilateral uniformity* observed. The aqueous humor,

Corneal opacities.

Appearance of.

Pupils.

iris, lens, and vitreous may be observed if the lens is moved or the eyes of the patient turned as becomes necessary.*

Adhesions, scars and iritis.

Iris.—The iris may show inflammation, adhesion (synechiæ), or the coloboma of a past iridectomy. *Iritis.* A delicate pink pericorneal injection maximal at the corneal margin and not moving on sliding pressure upon the conjunctiva, a darkened turgid greenish iris, photophobia, and a sluggish, contracted, sometimes irregular pupil, indicate iritis.

Simple vs. Specific.

Conjunctivitis.—Shows a vivid and tortuous general hyperæmia most marked in the cul de sac, the vessels being readily moved-by sliding pressure. Common in all catarrhal affections and eye-strain, it may in severer suppurative forms be gonorrheal, diphtheritic or meningeal, or, associated with pain, a symptom of corneal ulcer or even glaucoma. *Ecchymoses* occur as the result of traumatism, violent physical strain, asthma, epileptic seizure or in connection with hæmophilia, purpura, leukæmia, malignant endocarditis or pernicious anæmia.

Ecchymoses and apoplexy.

In several cases observed by the author an apparently causeless hemorrhage has preceded for weeks or months a cerebral apoplexy.

Light.

Red reflex.

Fugitive reflex, and refraction.

Retinoscopy.—Sitting one metre from the patient, whose vision should be directed straight ahead, the source of light being slightly higher than the head, a beam from the perforated mirror is thrown upon the pupil. The red reflex appears if the media are transparent. The mirror should then be tilted in all directions, the fugitive central reflex produced being carefully noted as it gives an exact clinical indication of the nature of the refraction.†

A subtraction of one diopter is always made for the one metre distance, or a plus one spherical lens may be placed in a trial frame before the eye, in which case the subtraction need not be made. The eye must be completely under the influence of a mydriatic in doing retinoscopy, if accuracy is desired.

* It is best to dilate the pupil for inspection of the lens margins and anterior layers of the vitreous. (Thorington.)

† If the source of light be vertical (argand burner, not electric light) and the mirror concave, a straight pupillary shadow moving quickly in a direction opposite the tilting of the mirror indicates either a normal, hypermetropic or less than one diopter myopic eye. Movement of the shadow in the same direction as the mirror tilt indicates myopia exceeding one diopter and the higher the error the more crescentic and slow moving is the shadow. Astigmatism is evident if the shadow edge moves differently or with different rapidity in opposed meridians. Simple astigmatism is present if there is movement of the shadow in only one meridian, the other being normal; compound astigmatism, if the movement of the shadow is similar in each meridian but of different degree. Mixed astigmatism is evidenced by different directions of the shadows in opposite meridian.

PLATE VI

Fig. 1 Albuminuric Retinitis of Pregnancy

Fig. 2. Retinitis Diabetica

Fig. 3. Retinitis Pigmentosa

Fig. 4. Detachment of the Retina

(From Thorington's "Ophthalmoscope and How to Use It")

DESCRIPTION OF PLATE VI.

FIG. 1.—ALBUMINURIC RETINITIS OF PREGNANCY. NEURO-RETINITIS. PAPILLO-RETINITIS. LEFT EYE. DIRECT METHOD

Fundus Changes. Disc hidden and slightly swollen. Punctate dots above and at the macula (neuritic dots) are conspicuous. Areas of exudation some distance from the disc. Hemorrhages numerous, small and flame-shaped. The veins full and tortuous, some are covered by the swollen retina.

FIG. 2.—RETINITIS DIABETICA. RIGHT EYE. DIRECT METHOD.

Fundus Changes. Arterio-sclerosis showing in the upper and lower temporal vessels where they cross. Small flame-shaped and round hemorrhages scattered irregularly in the fundus. The disc edges are foggy and the membrana cribrosa is indistinct. The intermediate zone and in fact the entire disc has a canary-yellow color appearance.

FIG. 3.—RETINITIS PIGMENTOSA. RIGHT EYE. DIRECT METHOD.

Fundus Changes. The periphery of the eye ground is characteristic of many myopic eyes, the choroidal vessels being very conspicuous. The retinal vessels are not very numerous and the smaller ones can be traced with difficulty in the periphery. The disc is quite yellow. The retinal vessels narrow. The reflex immediately around the disc approximates the normal, but beyond this the condition is atrophic. A few stellate pigment spots, together with irregular pigment massings on the vessels, are scattered throughout the fundus. This is an unusual variety of the disease. The patient is color blind for red and has night blindness.

FIG. 4.—PARTIAL DETACHMENT OF THE RETINA. RIGHT EYE. DIRECT METHOD.

Fundus Changes. Retina detached downward and forward. The wavy condition of the retina, the course of the dark-colored vessels without their usual light streaks, as they appear on the detachment, are all quite characteristic. This disc appears foggy because it is out of focus as compared with the detachment. The disc is seen with -7 D., whereas the detachment is best seen with a $+1$ D. The white streaks are very likely congenital and possibly obliterated vessels. A rupture of the choroid would be crescentic in shape and situated elsewhere in the fundus.

The Fundus Oculi.—*Indirect Methods of Ophthalmoscopy.* Proper dilatation having been secured,* the patient with head slightly bent forward sits close to the source of light which is on a level with the ear lobe.† The examiner sits from 1½ to 2 feet distant, the left shoulder advanced, a convex lens (13 diopters) in the left hand, the ophthalmoscope in the right. The perforated centre of the larger (concave) mirror should be exactly opposite the eye-hole of the instrument in which a + 3 diopter lens is placed. When a beam of light is thrown (from the mirror) upon the pupil a rosy reflex entirely fills it. The patient's gaze should be fixed upon the examiner's left ear if the left eye is under examination, upon the right little finger tip as extended while holding the ophthalmoscope if the right eye is under inspection. This reveals the optic disc, and slowly shifting the patient's gaze from right to left and up and down brings the periphery into view and the macula should be seen when the patient looks directly at the centre of the examiner's forehead. The large convex lens should be held between finger and thumb at about 2½–3 inches from the eye or at such distance as is indicated by its strength and the clearness of the image. It may be steadied by resting the little finger upon the patient's brow.‡ *In indirect ophthalmoscopy the image is inverted, all relations being exactly reversed but the image is large and the method easy.* Astigmatism is indicated by shrinkage or expansion of the image in any *meridian* attending the slow withdrawal of the lens, and in general the *contraction of the field upon withdrawal indicates hypermetropia; its enlargement, myopia,* so that simple, compound, mixed or irregular astigmatism may be thus detected. These conditions are merely suggestive, not accurate.

Dilating pupil.

Position of patient and examiner.

Red reflex.

Observing disc.

Accessory lens.

Gives inverted image.

Refractive errors revealed.

The Direct Method.—The advantages of this method lie in the magnified (cornea and crystalline lens) *direct* image obtained and the better showing of details though a smaller surface is brought into view in any one position. The light should be close to the patient but behind and slightly above the level of the ear corresponding to the eye under examination. The examiner must be able to *slowly* bring his own eye

Magnified direct image.

* A drop or two of 2% homatropine solution, or better, a fresh 4% cocaine or euphthalmin solution is dropped into the eye a half hour or more previously and followed after examination by a drop of eserine solution (gr. ¼ to 1 oz. of water).

† A light directly above the head may be used for this indirect method.

‡ Bed-ridden patients may be examined whether recumbent or sitting up by proper adjustment of the light and correct position on the part of the physician.

Technique.

within two inches of the one under examination and the eye used should correspond to the one examined (examiner's right, patient's right). The ophthalmoscope is applied flat to the cheek, the elbow at the side, the small mirror so adjusted as to give maximum reflection and the patient is told to look off into space over the shoulder of the examiner and

Relaxing accommodation.

under no circumstances to look at a near object (relaxation of accommodation). The examiner must relax his own accommodation or if necessary use a -1^{D} or -2^{D} lens* if the patient's refraction is normal. The proper lens must be inserted in the ophthalmoscope when the patient's refractive error does not permit a distinct view of the retinal structures and might lead to serious misinterpretation. If the examiner's refraction is faulty the proper glasses should always be

Use of mydriatics.

worn. As the patient must look directly in front of him to bring the macula into view and as the impingement of light upon that point will surely cause pupillary contraction, preliminary dilatation is often necessary.

MOTOR NERVES OF THE EYE.—*Motor Oculi* (3rd), *Patheticus* (4th), *Abducens* (6th). These three nerves closely related in origin and course and connected by an elaborate system of association fibres

Muscles supplied.

control the movements of the eye. *The 3rd supplies the ciliary muscle, levator palpebræ superioris, inferior oblique and superior, inferior and internal recti. The 4th supplies the superior oblique, the 6th the external rectus.*

Nuclei.

The *multiple nuclei of the 3rd* and *that of the 4th* lie beneath the floor of the Sylvian aqueduct, while *that of the abducens* lies lower beneath the floor of the 4th ventricle.

Often involved in basal lesions.

All are much exposed to pressure in basal lesions, *the 3rd emerging at the inner surfaces of the crura*, the *6th between the medulla and the pons*, while *the 4th is the only nerve emerging on the dorsal surface*, springing from the roof of the 4th ventricle immediately after it has *decussated* with its fellow. All three nerves may be affected in *gumma, meningitis, tumor, or hemorrhage*, by *syphilitic* or other forms of neuritis or by direct injury affecting the base, and *one may be affected alone*, the patheticus

Isolated paralysis of the 4th.

most rarely. Furthermore, *isolated paralysis of the 4th is indicative of cerebellar tumor or an exudate on the anterior inferior aspect of the cerebellum.* As the unilateral lesions of the base usually affect cranial nerves of the same side with the exception of the patheticus, while producing hæmiplegias of the opposite side, the value of crossed paralysis

*It is entirely unnecessary and wholly unwise to close one eye in using the ophthalmoscope and the observer should imagine that he is looking at a distant object.

PLATE VII

Fig. 1. Atrophy of the Optic Nerve (Post Papillitic Atrophy) Also Medullated Nerve-fibers

Fig. 2. Primary Optic Atrophy

Fig. 3. Retino-Choroiditis

Fig. 4. Glaucoma

(From Thorington's "Ophthalmoscope and How to Use It").

DESCRIPTION OF PLATE VII.

FIG. 1.—ATROPHY OF THE OPTIC NERVE (POST PAPILLITIC ATROPHY). ALSO MEDULLARY NERVE-FIBRES. RIGHT EYE. DIRECT METHOD.

Fundus Changes. Disc is bluish, the edges not well defined, lamina cribrosa not present. As a coincidence there are medullary nerve-fibres present. The whole fundus is mottled (map-like). The retina is atrophied and the macula cannot be distinguished. Arteries are straight and some of the larger veins slightly tortuous.

FIG. 2.—PRIMARY OPTIC ATROPHY. RIGHT EYE. DIRECT METHOD.

Fundus Changes. Disc bluish and glistening. Membrana cribrosa at centre of disc. A cilio-retinal vessel is seen on the temporal edge of disc passing toward the macula. Fundus reflex apparently normal. Vessels are not particularly narrowed at the present time. Anterio-sclerosis evident at crossing of vessels. Disc has "saucer" shaped excavation.

FIG. 3.—RETINO-CHOROIDITIS (SPECIFIC). LEFT EYE. DIRECT METHOD.

Fundus Changes. Nasal edge of disc hidden and cannot be distinguished from the neighboring retina. Temporal edge of disc is clear and reveals a narrow crescent. A few yellowish-colored spots seen in the choroid. Other spots of choroiditis have become absorbed and white areas (atrophy) have taken their places with irregular pigmentations. The choroidal circulation is exposed in the periphery. A large patch of retino-choroiditis is seen close to the temporal side of the disc.

FIG. 4.—GLAUCOMA. LEFT EYE. DIRECT METHOD.

Fundus Changes. Edge of disc seen with + 6 D. and the bottom of the cup is seen with a − 5 D. Vessels of the retina disappear as they pass into and around the edge of the disc, and are cut of focus when they reach the bottom of the cup, where they appear indistinctly at the nasal side. The nerve is bluish or pearly white in color and atrophic (glaucoma atrophy). The edges of the disc have a distinctly yellowish color and the pigment is broken into fine particles. There is a peculiar redness showing at the macula.

The cupping embraces the entire disc.

as a localizing symptom is readily seen. Partial paralysis of motor oculi innervation may result from lesions involving individual nuclei or the roots springing from them and passing through the knee of the internal capsule. *Complete uncomplicated unilateral paralysis of any motor ocular nerve indicates a lesion of its trunk and the effects of orbital and sinus lesions or pressure at the sphenoidal fissure should be held in mind.* Hemiplegia of the opposite side co-existing suggests:—In the case of the *motor oculi a midbrain lesion or a tumor involving pressure upon a crus* in which latter case the hemiplegia precedes the eye involvement. *If the paralyses are coincident* and *simultaneous a lesion above the corpora quadrigemina is suggested.*

Crossed paralysis.

Paralytic Symptoms.—*Motor Oculi. Abolition of pupillary reflex and fixed moderate dilatation* due to unopposed sympathetic influence, the *eyeball swings outward and slightly downward* (unopposed influence of abducens and patheticus) and any attempt to overcome the *ptosis* present is accompanied by excessive contraction of the occipito-frontalis and a characteristic predominance of forehead wrinkles on the affected side.* Irritant lesions will of course cause contracted pupils and various ocular spasms, such as nystagmus, nictitating or various non-rhythmic movements, blepharospasm or hippus, may replace ptosis.

Fixedly dilated pupil.

Strabismus and ptosis.

Irritative symptoms.

TESTS FOR LESIONS OF THE 3RD, 4TH AND 6TH NERVES.—These are essentially tests of pupillary activity (see page 552) and muscular strength, the latter being best indicated by diplopia (double vision) inasmuch as marked impairment may exist without strabismus (squint).

Strabismus.—*The "cross eyed" person cannot bring the visual axis of both eyes simultaneously to the same point* and ordinarily the defect is lateral, more rarely vertical. In a *divergent strabismus* the defective eye swings outward, in *convergent squint* inward. If the squint be spasmodic (*concomitant*) it is always made evident by asking the patient

Divergent vs. Convergent.

Concomitant.

* Firm downward pressure over the brow by cutting off the action of the frontalis enables one to estimate the actual degree of ptosis. Ptosis or pupillary signs without other symptoms of oculo-motor involvement are not infrequently seen in locomotor ataxia, as in a case under the author's observation for a long time. Ptosis may be incomplete and is simulated by hysterical orbicularis spasm, a transient ptosis upon waking seen in neurotic or over-worked persons, or by paralysis of the Müllerian fibres of the sympathetic in the orbital tissue, a paralysis which differs from true ptosis in the pupillary contraction and the symptoms of inflammation and edema of the corresponding lower lid. Furthermore, the inhibiting action of the association fibres of the trigeminus (5th) may cause ptosis in trigeminal lesions.

to look straight ahead, furthermore, the eye moves in any direction in direct ratio to the movement of the sound eye, though retaining its lack of parallelism, and, diplopia is usually absent. *Paralytic strabismus* on the other hand shows either total loss of movement in the affected muscles or marked weakness which, however may only be apparent when the patient looks in a certain direction or under continued fixation of the gaze upon one object in the weak axis. The abducens is commonly the nerve affected.

Paralytic.

Causes.

Diplopia.—*Double vision occurs only in paralytic squint except when developed by special tests.* It is a frequent symptom of the toxic irritation produced by certain drugs such as alcohol and belladonna or by any irritability or destructive lesion at the base of the brain. Having the patient follow with his eyes the finger or pencil moved laterally and vertically before them and their convergence as it is brought gradually close to his nose measures roughly the extent and indicates the specific muscles affected. He should be asked to tell the number of fingers held before him in each position, no rotation of the head being permitted. To find the eye and muscle producing double vision requires in many instances a resort to special tests.*

Simple tests.

Looking "toward" or "from" the lesion.

Conjugate Deviation.—*If both eyes deviate to the same side they are said to "look towards" the lesion, if it be paralytic,* and *"away from" it if the condition be spasmodic (irritation)* but *in lesions of the pons the reverse is true.*

Effect of lesions.

TRIFACIAL.—The 5th consists of two roots, one sensory and one motor. The 1st rises from a large pontine nucleus, the latter in a small one lying just internal to it, both being in the floor of the 4th ventricle. *Lesions of the nerve or its nuclei produce the following symptoms:*—**Ophthalmic Division.**—*Anæsthesia* of the conjunctiva, skin of brow and nasal bridge, the upper lid, forehead and anterior portion of scalp.

Taste affected.

Supra-maxillary.—Anæsthesia of the alæ nasi, cheeks, temples and nasal mucous membrane, upper pharynx, tonsils, soft palate, roof of mouth and upper teeth, upper lip; diminished taste and lost palate reflex. **Inferior Maxillary.**—Taste impaired in anterior portion of

* *The Candle Test.* In the usual test spectacle frame of the test case place in front of one eye one red glass; standing several metres away move about a lighted candle until two images (one red, one plain) are reported by the patient who also states where the colored image lies with relation to the other. Then the colored image represents the visual axis of the covered eye, the plain one that of the uncovered. If now by moving the candle in the direction of the colored image the double vision increases it proves the eye covered by the red glass to be affected and the weak muscle is that which turns the eye in the direction of the false image.

tongue, salivary flow checked; anæsthesia of the mucous membrane of the mouth, the lower part of the face and lower lips, trifacial neuralgia, paræsthesia, and, *as it receives the motor root*, trismus or paralysis of the muscles of mastication. Unless the paralysis be bilateral little actual disability occurs but in complete paralysis the jaw drops and even swallowing is difficult. Dryness of the nasal, buccal and conjunctival mucous membrane of course follows a lesion of the 5th and partial deafness may result.

Neuralgia.

Trismus or jaw drop.

Deafness perhaps.

Taste.—This sense is tested by placing upon the tongue successively, sugar, salt, quinine and tartaric acid, the tongue being protruded continuously throughout the procedure and the anterior and posterior portions separately tested. *Paræsthesia* (taste perversion) may be evident or admitted.

Substances used.

Motor Action.—The deviation of the jaw is toward a unilateral lesion, and when the teeth are clenched the affected muscle fails to contract forcibly under the palpating finger placed over the masseters. The perception of musical notes of different pitch tests the field of hearing.

Jaw deviation.

Tensor tympani.

Miscellaneous Symptoms.—No sneezing follows tickling the nose, irritating substances do not affect the tongue or nostrils, a cup placed to the lips feels as if broken, the patient may support the jaw with the hand to aid mastication and swallowing, and atrophy and loss of reactions may follow the paralysis. Herpes zoster, dental decay and ulceration of the nasal, buccal, and corneal surfaces may occur as expressions of a lost trophic function. Trismus is the result of infective tetanus or general convulsions, a convulsive tic occasionally results from reflex irritation. Tonic spasm also occurs, rarely, in hysteria and after long continued and enforced stretching of the muscles. Clonic spasms, slow or fast, are seen in the fear, chill and teeth grinding so common in children and indicative of over-wrought nerves, worms, gastro-intestinal disorders, adenoids, impacted cerumen, etc. The trifacial, while frequently the seat of neuralgia, is well protected from ordinary injuries and pressure; but may nevertheless be involved in certain bulbar palsies and in focal and gross lesions at the base.

Nasal and labial.

Masseters.

Trophic changes.

Spasm.

Neuralgia.

6TH NERVE.—Abducens.—This has the longest course of any cranial nerve and is frequently involved in basal lesions, producing internal strabismus. Its associations in disease are essentially the same as those of the 3d nerve.

THE 7TH NERVE.—The Facial.—*An accurate knowledge of the origin and connections of the facial nerve is of the utmost importance to*

Motor.

the clinician. It is almost purely motor and essentially the nerve of facial expression. The cortical fibres *decussate* in the raphé of the tegmentum and enter their nuclei at the junction of the pons and medulla, external to that of the 6th.

Origin and course.

From its origin in the pons nucleus it sweeps about the nucleus of the 6th, which lies just external, and *emerges* with the 8th nerve *between the restiform bodies and the olive at* which point *any lesion must include both nerves* (7th and 8th) as they run together into the internal auditory meatus. In the Fallopian aqueduct it runs with its branch to the stapedius and the fibres of the chorda tympani and emerging at the junction of the mastoid and ear it passes to the facial muscle. Though its cortical centre lies below the motor areas of both leg and arm (inferior portion anterior frontal convolution) it lies between the two in the internal capsule, crus and pons.

Association.

Cortical centre.

FACIAL SPASM AND PARALYSIS.—It is evident that *a lesion may be either supranuclear, nuclear, or infranuclear,* and that the last may be (a) *within the aqueduct,* (b) *external.* In *supranuclear* paralysis usually associated with hæmiplegia the orbicularis is not affected, R. D. is absent and the paralysis is on the same side as that of the leg or arm muscles, save in lower pontine lesions. *Nuclear* paralysis is essentially that of the infranuclear type, but seldom exists alone. *A lesion within the aqueduct* involves the chorda tympani and the stapedius branch, hence *there is loss of taste over the anterior ⅔ of the tongue,* and often hyperacusis (exaggerated sound sensation). *A lesion involving the nerve at its point of emergence* involves the auditory, hence is associated with the various symptoms of irritation or paralysis of that nerve. If the fibres are involved between the nuclei and their decussation (lower pontine lesions) *crossed* paralysis results.

Three varieties.

Supranuclear.

Nuclear and infranuclear.

General irritative symptoms may be present, muscle spasm, blepharospasm and convulsive tic, *symptomless paralysis* of such muscles as the stylohyoid, or *characteristic paralysis of the muscles of expression* in which case *facial expression lines are obliterated* and *the victim cannot whistle, draw the corners of the mouth outward, close the watering eyes tightly or wrinkle the forehead of the affected side. Bilateral* palsy is rare as an independent lesion. Monoplegia is the type and in hæmiplegia with alternating facial paralysis we deal with the nuclear form.

Usual type.

AUDITORY NERVE.—The dorsal root contains the auditory fibres, the ventral those of equilibration and passing out on either side of the restiform body they join and within the internal meatus pass to the cochlea for audition and to the vestibule and semicircular canal for

Audition and equilibration.

equilibration. The cortical centre for hearing lies in the opposite temporal lobe, that of equilibrium in the cerebellum. Cortical centres.

Symptoms.—*Deafness and paræsthesia if the cochlea only be involved. Vertigo, vomiting, tinnitus aurium, sibilant or roaring sounds* ("escaping steam") associated with *disturbance of equilibrium* if the labyrinth branch be affected (Ménière's syndrome). That common and ordinarily trivial symptom, *tinnitus aurium*, like deafness, may be due to many causes, such as catarrh of the middle ear and Eustachian tube, or neurasthenia (the common causes), anæmia, digestive disorders and various toxæmias, inflammation or impacted cerumen in the external meatus, tympanites, meningitis, syphilis, brain tumor, full doses of salicylate, quinine, etc. *Tinnitus cerebri*, i.e., the sound seeming to arise in the brain itself, is frequent in arterio-sclerosis and is also found in meningeal cases. Cochlea. Ménière's syndrome. Tinnitus aurium.

Deafness.—"*Nervous deafness*," *i.e., that arising from cortical, nuclear, or peripheral nerve lesions is rare; that due to catarrh of the middle ear and impacted cerumen common.* Hearing is ordinarily tested by the watch, which should be held at varying distances from the ear and the range of the patient compared with the known normal for the particular watch used. If deafness is evident the tuning fork should be employed to test by bone conduction the integrity of the cochlear nerve and centres. If the vibrating fork or watch is best heard when placed directly upon the mastoid the *nerve* is not at fault.* In most instances *sudden* deafness is due to impacted cerumen, hysteria, or syphilis of the internal ear. Naso-pharyngeal catarrh, usually associated with Eustachian involvement, adenoids and polypi must be remembered as well as growths and swellings in the auditory meatus. In headache, mental strain or fatigue, or pronounced mental irritability, sounds may be exaggerated (hyperacusis) or actually painful. In hysteria, decided or complete deafness may come on suddenly, is manifested chiefly in defective range for notes of varying pitch, and *shows relatively equal loss of both aerial and bone conduction.* It may suddenly disappear and usually follows emotional shock. Nervous form rare. Tests. Sudden deafness. Ordinary form. Hyperacusis. Hysteria.

Meniere's Disease.—This combines progressive nervous deafness with paroxysmal attacks of vertigo, forced movements, tinnitus aurium and a peculiar inco-ordination of the eye and head muscles. Nausea.

* Rinné's test:—Set tuning fork in vibration, place handle against mastoid or zygoma, closing meatus at same time. When it becomes inaudible, open the meatus and hold the still vibrating fork to it. Normally the sound should still be heard at the meatus; abnormally bone conduction is the greater.

vomiting and even transient unconsciousness may accompany the attacks.

Word deafness.

Cortical Lesions.—Though rarely bilateral, cortical lesions may be present and a lesion of the left cortical centre may cause word deafness, i.e. inability to interpret the sounds heard. Tumor, hemorrhage, meningitis, abscess, and syphilis may involve the internal capsule or corpora quadrigemina. The nerve itself may be injured or compressed by tumors and inflammatory or hemorrhagic exudates, or be involved in diphtheritic paralysis, locomotor ataxia, and traumatism. Deaf mutism is not an unusual sequence of cerebro-spinal meningitis. *Labyrinthine* disease is usually a primary disease *of the labyrinth or secondary to middle ear suppuration and necrosis and mastoid disease.* Certain forms of, usually transient, deafness follow the administration of quinine and the salicylates, violent concussion or constant and repeated l. ser sounds (occupational deafness).

Deaf mutism.

Transient deafness.

Seldom paralyzed alone.

GLOSSO-PHARYNGEAL NERVE.—The 9th nerve is seldom paralyzed alone and a lesion is recognized only through its sensory disturbances, though it is motor for the stylo-pharyngeus and middle pharyngeal constrictors. The sensory fibres run to the brain with the 5th nerve and a loss of taste over the posterior surface of the tongue, and of the pharyngeal reflex are the only sources of information. Root involvement does not affect taste on account of the peculiar course of the sensory fibres.

A marvelous nerve.

Spinal accessory association.

Dysphagia.

THE VAGUS.—The 10th nerve has most interesting and complicated functions. It is *motor for the pharynx, larynx and soft palate and both motor and sensory for the heart, respiratory passages* and *most of the abdominal viscera,* in connection with which the sympathetic plays an important part. Its motor fibres, including the cardio-inhibitory group, are contained in what was formerly regarded as the bulbar porton of the spinal accessory which joins it outside the cranium. The pharyngeal plexus is formed by the vagus and glosso-pharyngeal nerves and supplies the pharyngeal muscles and mucosa, hence a lesion of the nuclei or trunks gives rise to marked dysphagia.

Palatal paralysis.

Tests.—Complete paralysis of the palate is indicated by regurgitation of fluids through the nose in swallowing and thick nasal speech. If the palate be examined while the patient says "eh" and "ah", defective upward movement is perceived; if unilateral, immobility of one side with dragging of the median raphé towards the sound side.

Laryngeal supply.

The Laryngeal Nerves.—The superior and inferior laryngeal nerves supply the musculature and mucosa of the larynx. The recur-

rent laryngeal which supplies the mucosa of the lower portion and all muscles except the crico thyroid, is especially liable to pressure paralysis by reason of its relation to mediastinal structures, aneurism being one of the commoner causes of paralysis of the left recurrent. Bilateral paralysis of the abductors causes imperfect approximation and separation of the vocal cords in respiration and voice production. The symptoms are inspiratory stridor without aphonia, the cords being nearly approximated. It is encountered in cases of hysteria and occasionally follows a simple laryngitis though usually toxic. Pressure upon one recurrent nerve, usually aneurismal or due to pleural adhesion, produces unilateral abductor paralysis, immobility of the affected cord in inspiration and hoarseness. Adductor paralysis is usually hysterical and the separation of the cords produces aphonia. (See fig. 217.)

Chiefly recurrent branch.

Laryngeal Spasm.—The croup of children, locomotor ataxia (laryngeal crisis) and hysteria are the commonest sources, the last form being chronic and usually associated with speech production.

Common types.

Anæsthesia.—Due to diphtheria, neuritis, bulbar paralysis, or hysteria, makes feeding difficult and inhalation pneumonia common. **Hyperæsthesia** is rare, but occasionally seen, especially in sexual neurasthenia.

Dangers.

Cardiac Plexus.—Formed by the vagi and sympathetic nerves its branches control the activity of the heart. Stimulation of the vagus slows or temporarily arrests the heart action, and in rare instances voluntary inhibition has been observed. In complete paralysis the accelerator fibres act unhindered causing rapid heart action. Palpitation and cardiac pain are probably appreciated through the vagus path.

As affecting heart action.

Gastric Branches.—The vagi control the activity of both stomach and esophagus, conveying sensory impressions as well as motor stimuli and are probably concerned with so-called nervous dyspepsia and the gastric crises of locomotor ataxia. In bulbar paralysis the nuclei of the accessory nerve and the hypo-glossal are involved with those of the vagus.

Bulbar paralysis.

SPINAL ACCESSORY.—The chief function of the 11th nerve is the supply of the sterno mastoid and trapezius muscles, and, through the vagus, the pharynx and larynx.

Sterno mastoid and trapezius.

Tests.—The laryngeal paralyses have already been covered. Paralysis of the sterno mastoid prevents wholly or in part rotation of the chin towards the sound side, and if the trapezius be paralyzed, shrugging of the shoulders is interfered with. (See also p. 546.)

HYPOGLOSSAL NERVE.—The 12th is entirely motor and supplies the tongue and depressors of the hyoid bone. It rises from a nucleus nearly median in the lower portion of the floor of the 4th ventricle and its cortical centre lies in the lower frontal convolution.

Cortical vs. Peripheral palsy.

Tests.—Note evidence of wasting and tremor or fibrillary contraction. Wasting indicates a nuclear or trunk lesion, its absence a supra-nuclear or cortical lesion. Deflection of the tongue towards the paralyzed side is evident on protrusion. (See also p. 544.)

THE SYMPATHETIC NERVOUS SYSTEM.—The anterior horns of the cord carry fibres of unknown cortical origin to the sympathetic ganglia through a special set of axones running in the anterior nerve roots, and sympathetic ganglion cells with their axones in turn constitute the motor sympathetic neurone. A similar set of fibres (sensory) traverse the posterior roots and there is a free communication between the cranial nerves and the sympathetic system. The vaso-motor fibres are especially plentiful in the anterior roots of the dorsal region. The visceral fibres are chiefly in the cervical region where they accompany the accessory and vagus nerves and ultimately pass to the alimentary canal.

The wonderful automatic control exercised by these nerves cannot be dealt with here but though their activity may continue when all central nervous connections are cut, they are to a certain extent controlled and regulated by the higher centres, The cilio-spinal fibres are important in diagnosis, irritation causing pupillary dilatation; section, contraction. Other symptoms of sympathetic origin are:—vaso-motor spasm or relaxation, abnormal sweating in the involved areas and various disturbances of glandular secretion and cardiac and visceral activity.

DISEASES OF THE BRAIN AND SPINAL CORD.

HEMORRHAGIC PACHYMENINGITIS (*internal meningeal hæmatoma*).—This may be encountered in either the brain or the cord. The *cerebral form* is almost exclusively confined to the insane (especially paralytic dementia) and the aged, though occasionally met with in vascular degeneration, profound anæmias, alcoholisms, cachexias, diseases of the purpuric type and even in acute infections. *Pathologically* three forms may be seen, (a), *simple subdural hemorrhagic exudate*, (b) *an inflammatory exudate converted into a delicate vascular membrane by granular tissue and vascular extension*, (c) *the two conditions combined.* In the insane and aged it is supposed that the cerebral atrophy so frequently encountered *is a contributive* factor.

Chiefly in the insane, alcholic and aged.

Three forms.

Symptoms.—If any exist they are those of intracranial pressure, recurrent convulsions, coma and paralyses.

SPINAL FORM.—This may involve any portion of the dura mater of the cord (most commonly the cervical enlargement) or even be accompanied by cerebral hæmatoma. *Pressure symptoms* occur accompanied by secondary degeneration and producing intense peripheral neuralgic pain chiefly brachial and cervical with hyperæsthesia and paræsthesia, slowly progressive muscular weakness, paralysis and muscle atrophy. **Prognosis.**—The disease is chronic, usually fatal within two years, and during the later months is often associated with *spastic* paraplegia of the lower extremities.

Pressure symptoms.

Chronic course.

Differential Diagnosis.—It cannot be distinguished from *tumors of the cord. Amyotrophic lateral sclerosis* lacks the severer neuralgic pain, and *syringomyelia* may be differentiated by its characteristic sensory symptoms. See p. 600. Pseudo-membranous and purulent pachymeningitis are not worthy of detailed consideration.

EXTERNAL PACHYMENINGITIS.—Both the cerebral and spinal forms *yield the symptoms of compression* and ordinarily result from traumatism, caries or the extension of some suppurative process. It is most frequently secondary to syphilis and tuberculosis, tumors or suppuration, involving the adjacent structures.

Hæmatorrhachis.

Extra-meningeal Hemorrhage.—Aneurismal rupture or traumatism involving the peridural plexus may cause hemorrhage usually with pressure symptoms. **Intra-meningeal Hemorrhage** is more often a complication of acute febrile toxæmia, convulsive seizures and the rupture of basilar vertebral aneurisms, less often traumatism.

Symptoms.—These are usually *localized pressure symptoms* of sudden development, and cases secondary to acute febrile toxæmia and traumatism furnish the most favorable prognosis.

Hæmatomyelia.—Hemorrhage into the cord itself, *usually associated with hæmatorrhachis* may result from the causes of intra-meningeal hemorrhage, but is more often due to direct injury and *is undoubtedly one of the causes of the disturbances noted after injuries involving violent flexion or torsion of the spine.* The hemorrhage varies greatly in extent and position and the paralysis may be paraplegic or unequally affect the two sides, but is frequently unilateral producing the well known Brown-Sequard symptom complex, viz.:—unilateral motor paralysis with anæsthesia of the opposite side, etc. (See p. 526.) Pain is usually slight or absent; paræsthesias are common and the lumbar or cervical regions are most frequently affected. *The onset is sudden,* and in

Usually directly follows injury.

Sudden onset valuable sign.

low seated lesions urinary retention or incontinence is often observed.

Prognosis.—Partial recovery is the rule and fatal termination the exception, both primary and residual paralyses are chiefly of the spastic type.

CAISSON DISEASE.—This obscure ailment is encountered in submarine workers exposed to high grades of atmospheric pressure and seems to be connected with the sudden relief of such pressure. The changes seem to be strictly limited to the cord, taking the form of punctate hemorrhage, myelitis or laceration. There is an unproven theory that it is due to the release of nitrogen taken up by the blood under high pressure.

Decompression *the* factor.

Symptoms.—Immediately after leaving the caisson or perhaps not for 48 or 72 hours a series of symptoms develop varying in severity from excruciating pain in the lower extremities to spastic paraplegia with anæsthesia and urinary retention. Coma is sometimes observed, but the upper extremities are seldom affected and many minor symptoms such as temporary albuminuria, vertigo, deafness and tinnitus aurium may be encountered. The disease is rarely fatal and the symptoms ordinarily disappear in a few days or weeks.

Symptoms variable.

Transient.

"MILIARY" ("DIFFUSE", "TUBEROUS") **SCLEROSIS** may be dismissed as of little practical importance, all three varieties being associated with mental disease and seldom encountered save in the autopsy rooms of asylums.

Unimportant.

MULTIPLE SCLEROSIS ("insular," "disseminated") is a disease of unknown causation occurring chiefly in young male adults and characterized anatomically by an extensive irregular dissemination of reddish gray areas of sclerosis involving both the gray and white substance of all portions of the cerebro-spinal tract. The cerebral lesions ordinarily predominate and in the cord the cervical region and the cauda equina are usually chiefly affected. In the former the brain stem and basal ganglia suffer most.

Male adults chiefly.

Whole tract involved.

Diagnosis.—*Intention tremor, lessened in recumbency, nystagmus, scauning speech and transient palsies are the important symptoms.* Vertigo, optic atrophy, spastic weakness of the lower extremities and stiff awkward gait is common, enfeebled mentality and sudden attacks of coma may occur and *the symptoms may be extraordinarily varied and slow and insidious in development.* The sphincters may be affected in the terminal stage. *Differential points. Paralysis agitans* lacks nystagmus and shows tremor during rest. *Hysteria* lacks nystagmus and pre-

Characteristic complex.

sents usually distinctive stigmata. **Prognosis.**—It is markedly chronic and terminates by intercurrent disease, usually after the patient has become bed-ridden. Marked chronicity.

CEREBRAL CONGESTION.—*Cerebral congestion has lost its place as an important clinical entity, most of the symptoms formerly classed under that head being due to toxæmia rather than mere congestion* and to be referred to some underlying disease to which congestion is purely secondary. Mental excitement, indeed any mental effort means temporary congestion and certain drugs such as alcohol, quinine and strychnia undoubtedly produce it. Passive hyperæmia must occur in certain obstructive conditions affecting the circulations, either of the brain itself, or of the body generally, as is seen in cardiac weakness of extreme degree from any cause. *The symptoms* are *headache of varying degree, often associated with a subjective sensation of pressure and pulsation, vertigo, tinnitus aurium, mental irritability and impaired concentration.* Reduced in importance. Familiar symptoms.

CEREBRAL ANÆMIA.—Cerebral anæmia may be (a) simply a part of general anæmia; (b) due to temporary vaso-motor changes; (c) the actual loss of blood, or (d) arterio-sclerosis. *Nausea, vertigo, faintness or actual syncope are the symptoms of the first three groups; mental depression, impaired concentration, drowsiness during the day and particularly after meals, frequent headache and insomnia the chief symptoms of the last group, but there is no sharp line of division.* The headaches associated with marked general anæmia are usually dull but may assume a very violent type. The *insomnia* usually comes on after a period of sleep, the patient lying awake the balance of the night or merely catching short naps. Symptoms common to all types. Insomnia.

CEREBRAL EDEMA.—This condition is chiefly of interest in connection with the uræmia of Bright's disease, the unilateral paralysis or spasm sometimes observed therein being probably due to a localized cerebral edema. Congestive edema may be a part of extreme venous congestion or a local congestion due to obstruction, cerebral growths or abscesses. Presumptive only. Ante-mortem.

THROMBOSIS OF THE CEREBRAL VEINS AND SINUSES.—This condition is extremely rare as a primary lesion but much more common as a secondary event in other diseases. The existence of such a primary disease as suppurative otitis media, associated pulmonary infarct and marked cerebral symptoms resembling abscess, are the chief factors in diagnosis. Permits tentative diagnosis.

MENINGITIS.—Inasmuch as the characteristic symptoms of menin-

A lepto-meningitis.

gitis are very similar in all its acute forms these will be considered under one head. The lesion is in every case essentially an inflammation of the pia mater, localized or general, and shows a predilection for certain regions in its different forms. We distinguish (1). *Epidemic cerebro-spinal meningitis* due to the meningococcus of Weichselbaum. (2). *The simple or sporadic form* which is probably due to the same germ. (3). *Tuberculous meningitis* due to direct tuberculous invasion and associated in 80% of all cases with a pre-existing tubercular focus in other portions of the body which may only be evident upon a painstaking postmortem investigation. (4). *Syphilitic meningitis.* (5). *Secondary meningitis* due to septic infection or the toxins of acute infectious diseases. With the exception of the *syphilitic and secondary forms* the disease shows a special tendency to develop in *infancy and early childhood.*

Five varieties.

Young children most affected.

Morbid Anatomy.—In the *epidemic form* the entire pia mater of both brain and spinal cord is involved but to the ordinary symptoms of meningeal inflammation are added those indicative of an acute febrile toxæmia. In the *septic* and *secondary* forms, the pia of the convexity is chiefly affected often in limited areas *and the base may entirely escape, this being especially true of cases due to direct injury or extension of disease from the mastoid cells or nasal structures.* In *tuberculous meningitis* the involvement is so often *limited to the base* as to give it the name of "basilar meningitis," though it would seem that syphilitic meningitis was quite as well entitled to the term.

(1). Involves both brain and cord.

(5). Chiefly the convexity.

(3 and 4). Base chiefly.

In *cerebro-spinal meningitis* the general inflammation of the meninges of the brain and cord is associated with an exudation of serum, fibrin and pus, most abundant at the base of the brain and posterior surface of the cord though in malignant rapidly fatal cases nothing may be found but an intense acute congestion. In those of long duration there is thickening of the meninges and yellow areas mark the seat of past exudation. The ventricles contain turbid fluid, and in chronic cases, may be greatly distended. Foci of purulent infiltration and of hemorrhage may be found, and *cranial and spinal nerves* are of course enveloped by the exudate which constantly contains the *diplococcus.** In some cases the central canal is dilated. *In purulent meningitis* there is a tendency to the formation of multiple abscesses and the exudate will

(1). Morbid anatomy.

(5). Morbid anatomy.

* This germ described by Weichselbaum in 1887 is aërobic, non-motile, occurs in pairs, fours or short chains and unlike the pneumonococcus does not grow on gelatine but on blood serum, and shows small circular smooth colonies.

yield cultures of the associated germs. It is said that actinomycosis is indicated by yellowish granules, pyogenic cocci by a cohesive exudate, and that it is thick and yellow in tuberculosis. In chronic inflammation of the pia there is an increase in connective tissue producing linear surface projections and a marked tendency to adhesions. By compression cerebral nerves may be destroyed, ventricular openings obliterated and hydrocephalus produced. *Tuberculous Meningitis.* The development of miliary tubercles is superadded to an inflammation with sero-fibrinous or more commonly purulent exudate. The tiny gray points appear chiefly in the choroid plexus, along the Sylvian artery, inner surface of the dura, membranes of the cord, and even the retina. The usual changes, i.e., hyaline and caseous degeneration, occur and tubercle bacilli are ordinarily readily found. (3). Morbid anatomy.

Fig. 225.—Diplococcus intracellularis meningitidis. (Weichselbaum.) In actual specimens the germs are (like the gonococcus) chiefly within the polynuclear leucocytes.

Syphilis is indicated by the presence of soft grayish red areas, most common at the base, tending to undergo caseous degeneration, absorption and cicatricial contraction. Both the dura and the cortex may be involved. (4). Morbid anatomy.

Hydrocephalus results from an arrest of the flow of the cerebro-spinal fluid secreted by the choroid plexuses of the lateral ventricles by blocking, compression or adhesion, of the aqueduct of Sylvius or the foramen of Monro. In many cases of tuberculosis and practically all cases of syphilis the exudate is non-purulent. Hydrocephalus.

SYMPTOMS OF THE VARIOUS TYPES OF MENINGITIS.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.—(Petechial fever, spotted fever, malignant purpuric fever.) The *incubation* period is *undetermined*, the *onset* usually *sudden*, though *headache, anorexia* and *pain in the back may precede it.* There are *chill, causeless projectile or regurgitant vomiting, headache, severe backache, painful cervical and dorsal rigidity and usually moderate fever* (101°–102° F.). *Auditory and visual hyperæsthesia co-exists with restlessness, irritability and muscular tremor. The head is retracted* (boring occiput), *opisthotonos* or more commonly *orthotonos* may occur and in children, general *convulsions. Facial spasm, strabismus and ptosis* are common and pain in the head, back and extremities increases, and less frequently the trunk muscles may be paralyzed. *Spinal or general hyperæsthesia* is often noted and marked *irritability* may give place to *delirium, stupor* and *finally* Irritative symptoms. Paralytic stage.

Urine. Fever. Pulse and respiration. Blood. Constipation. Skin rashes.

coma. *Albumen*, *sugar*, and in malignant cases *blood* may be present in the urine. The *fever curve is extremely variable*, some cases showing hyperpyrexia, others almost no fever. *Respiration* is not markedly increased but the pulse is sometimes remarkably slowed and almost always weak, but in children may be much accelerated. *Leucocytosis is present in all cases* (20,000 to 40,000).* The bowels are usually *constipated*, the spleen moderately enlarged, excessive vomiting is an unusual but troublesome complication. The cutaneous symptoms vary greatly, *herpes is almost constant, purpuric spots occur in from two-thirds to three-fourths of the cases.* Dusky mottling, erythema, rose spots, urticaria and various other eruptions may be present.

Malignant cases may kill so promptly that the brain shows at autopsy only an acute congestion, they are often apyretic and are almost invariably associated with purpuric rash and feeble pulse, somnolence and pronounced æsthenia. **Intermittent Form.**—This is characterized by a febrile curve of a distinctly pyæmic type. A **chronic form** *may succeed* the acute. **Abortive Form.**—This somewhat doubtful type is characterized by a severe onset, an extremely short duration and a sudden termination, followed by rapid convalescence. Certain other mild types present symptoms hardly more than suggestive which promptly subside and in each of these varieties there is abundant opportunity for erroneous conclusions.

Errors common.

Complications.—These are, chiefly, pneumonia, often of a true meningococcus type, septic arthritis, peri- and endo-carditis and parotitis. Convalescence may be greatly prolonged and complicated by persistence of headache, nervous irritability or even mental impairment, or long persisting cranial nerve paralysis. Optic neuritis occasionally occurs as does parotitis, purulent conjunctivitis, choroiditis and iritis. Permanent deafness and persistent ocular palsies are very common and the frequency of nasal catarrh suggests the nasal secretion as a possible chief vehicle of infection.

Convalescence tedious.

Residual lesions.

Diagnosis.—*The presence of an epidemic, and the characteristic symptoms of cerebro-spinal irritation and compression* ordinarily make the diagnosis easy, yet the cerebral symptoms present in other acute febrile conditions are often misleading. *Tuberculous meningitis* usually lacks the sudden onset and rapid development. The rash, if present, is of great assistance; herpes, leucocytosis and diminished urinary chlorides assist in the differentiation from typhoid; the physical

Chief features.

Tuberculous form less abrupt.

* Leucocytosis is also present in tuberculous cases though not so constantly.

signs from pneumonia, though either disease may co-exist with meningitis. *The two most valuable and important symptoms of meningitis are Kernig's sign* and *lumbar puncture.*

Two best signs.

Kernig's Sign.—*Flex the thigh at right angles to the abdomen, the leg upon the thigh; if meningitis* is present, *extension of the leg is then prevented by contraction of the hamstrings. This sign is present in 90% of the cases,* but is also to be found in sciatica, diseases of the hip and knee joint and lesions of the cerebellum or upper motor neurones and to a less degree in old age or after prolonged fixed recumbency.

Exact procedure necessary.

Present in other conditions.

Fig. 226.—Kernig's sign. Proper method, i. e. preliminary flexion of thighs on abdomen followed by attempted extension of leg on thigh.

Lumbar Puncture.—Place the patient on the side, the body so bent as to get the *maximum dorsal curve,* carefully count the vertebræ and introduce the sterile needle of an aspirating syringe slightly to one side of the median line *between the 2nd and 3rd or 3rd and 4th lumbar vertebræ,* the needle point being directed towards the centre of the canal.* In *children,* a point midway between two spinous processes should be chosen, in *adults* it should pass just below the lower margin of the upper process of the chosen space. *A long needle is needed,* the distance traversed varying from 2 cm. in children to from 4–6 cm. in the adult.

Technique.

Point of election.

Children vs. Adults.

Depth of puncture.

Pressure is indicated by the force of the flow and normally represents 5–7 mm. of mercury, which may be increased to from 15–60 mm. in meningitis and tumors (Sahli). It is easily measured by attaching a small (1 mm. calibre) mercury manometer. Normally the fluid is clear and limpid, sp. gr.–1003, and but slightly albuminous. Marked cloudiness and the presence of leucocytes points to septic meningitis, the fluid being relatively clear in tuberculous meningitis and cerebral abscess. High albumin content is also significant though occasionally encountered in tumor and apoplexy. A smear should be made and stained for the specific diplococcus after centrifugalization. (See also p. 573.)

Pressure indications.

Fig. 227.—Kernig's sign. Improper method lacking the essential preliminary flexion of thighs upon abdomen. (After Sahli-Wiener.)

Cyto-diagnosis.

Diplococcus.

*To avoid the spinal cord Sahli prefers the interval between the 5th lumbar and the sacrum.

Prognosis.—Over 50% of the deaths occur in the first week and the mortality ranges from 20 to 80%, being higher in children than in adults. Hyperpyrexia, coma, repeated convulsions and sudden fall of fever are unfavorable signs.

ACUTE TUBERCULOUS MENINGITIS.—*The symptoms of this ailment are in general exactly the same as in other forms of meningitis, but as assisting differential diagnosis, there is frequently a history of pre-existing tuberculosis in other parts of the body, or the disease may be a part of an acute miliary process. Furthermore, there are usually marked prodromal symptoms* such as *general failure of health extending over several weeks, loss of weight and* appetite, *irritability*, headache *and restlessness.* The *actual onset* may be somewhat abrupt in children and there is the curious whining hydrocephalic cry,* apparently due to exacerbation of the headache. The pulse, at first rapid, becomes slow, and irregular, the respirations irregular and sighing and the *pupils are generally contracted* (stage of irritation). *In the second stage* there is *obstinate constipation, marked emaciation, retraction of the abdomen* (scaphoid belly), *boring occiput, stupor and irregular or dilated pupils, often associated with strabismus.* The *temperature* is usually moderate in both stages (101 –103° F). *The third stage* (paralysis, coma):—*Paralysis of the ocular muscles* is common, *optic neuritis* may be present, *diarrhœa may replace constipation,* and the victim lies with partially open eyes and dilated pupils, *in a typhoid state.* Temperature may at this stage be subnormal, the pulse rapid, and there may be antemortem hyperpyrexia. *The duration varies from a few days to a month, from two to three weeks being the common period.* Occasionally instances of a chronic course are encountered some of which lack the pronounced initial symptoms.

Pre-existing disease.

Prodromata marked.

1st Stage.

2d Stage.

3d Stage.

Typhoid *state.*

Duration.

General Comment.—The greatest diversity of symptomatology must be expected in a lesion involving the brain stem and perhaps the cortex in an inflammation associated with the outpouring of an exudate. The pupillary symptoms are quite constant as regards miosis of the early stage and the irregularity and dilatation of the later periods. Projectile vomiting and convulsive seizures or even tetanic contractions or catalepsy may be encountered in the earlier periods, and ocular palsies may exist alone or be associated with extensive cranial involvement.

Symptom diversity a feature of meningitis.

Ophthalmoplegia.

Prognosis.—Practically all cases die.

SYPHILITIC MENINGITIS.—*The symptoms are those of brain tumor, headache being pronounced and usually increased at night, and irritability, change in disposition and lack of appetite* are prominent

Brain tumor symptoms.

* Occasionally screaming is marked and almost constant.

features. *Causeless vomiting and vertigo* may be present and *eye symptoms or facial paralysis* may indicate involvement of the cranial nerves. *Cerebellar ataxia* and *polyuria* may occur *and if the Sylvian fissure be involved* there may be *hæmiplegia or aphasia.* To name all of the symptoms would be to review the whole subject of brain symptomatology. *The essential feature is the distinctly slow onset and the ready response of this condition to treatment,* the diagnosis being usually assisted by a history of syphilis or visible evidence of previous infection. *Without antisyphilitic treatment the diagnosis between syphilitic disease and brain tumor can hardly be made.*

Wide range of symptoms.

Slow onset and therapeutic test *the* features

ALCOHOLIC MENINGITIS.—The form of meningitis known as *wet brain* deserves special mention because of *its frequent occurrence in alcoholics* and certain peculiarities in its course and symptomatology. It usually terminates a long debauch and is frequently a terminal event in delirium tremens. *Its characteristics are stupor or coma with low muttering delirium, hallucinations and delusions.* There is a *general muscular tenderness, cutaneous hyperæsthesia, contraction of the pupils,* and after a short time *muscular rigidity, occipital retraction, loss of control of the sphincters, exaggerated reflexes and scaphoid belly.* The *temperature is variable,* often being normal or slightly raised throughout the disease, occasionally higher, especially at the close. The final stage may never be reached, the case terminating in recovery. The diagnosis is based largely upon the peculiar delirium and the history of alcoholism and is sometimes assisted by a knowledge of the occupation.

History of alcoholism.

Hallucinations marked.

Occupation may be suggestive.

SECONDARY AND SEPTIC MENINGITIS.—*These* cases present merely *the symptoms of meningitis* as already described, *usually in the presence of a suggestive antecedent acute or chronic disease, or the history of traumatism, accidental or operative.* In acute diseases error is likely to occur from two sources, one, the fact that certain acute febrile ailments such as pneumonia may present marked meningeal symptoms without true meningitis, the other, the liability that true meningeal symptoms may be masked by those of the disease which it complicates. These difficulties apply of course only to the stage of irritation and the doubtful diagnosis may almost always be made certain by lumbar puncture.

Primary disease suggests it.

Lumbar puncture in doubtful cases.

INFANTILE MENINGITIS.—A word is necessary concerning the special symptoms present in cases of *simple meningitis affecting babes.* The *period of maximum susceptibility* seems to lie between the third and sixth month. *The symptoms* are usually *sudden in onset, and frank*

Suggests cerebro-spinal fever.

in their character, pain in the early stage *being indicated by pulling the hair and constant crying or screaming. Emaciation is peculiarly rapid,* and *hydrocephalus is usually evident by the second week,* the head enlarging and assuming the typical outline, the fontanels bulging, and *changes in the eyes,* such as undue prominence, strabismus, and the usual pupillary symptoms being evident. *The tendency in the simple cases is to chronicity,* and recovery occurs in perhaps 20%. Many serious and permanent impairments may persist, these being chiefly deafness, mental defects and blindness.

CHRONIC MENINGITIS.—The symptoms of this ailment are essentially those of chronic syphilis or tubercular disease of the meninges and therefore consist almost wholly of pressure phenomena; any form of meningitis may however become chronic.

Pressure symptoms.

CONGENITAL HYDROCEPHALUS.—These cases cause much difficulty in labors and the children usually die within the first four or five years, rarely, even extreme degrees do not prevent full mentality and reasonably long life; but ordinarily, children are backward, mentally defective, and subject to spastic weakness of the lower extremities and perhaps convulsive seizures.

Course and termination.

HYDROCEPHALUS IN ADULTS.—This is ordinarily secondary to tumor though possibly in rare instances primary. There may or may not be enlargement of the skull through separation of the sutures, the symptoms are variable and the disease is not positively diagnosticable. During life the most common symptoms are headache, recurring attacks of coma, optic neuritis and occasionally, ataxia.

TUMORS OF THE BRAIN.—Varieties.—About ⅓ of tumors of the brain are tuberculous, and of these 80% occur in children, whereas cysts occur almost exclusively in adults. Sarcomata, usually primary, constitute about 20% of which 70% occur in adults; carcinomata of the softer sort and usually secondary 7%, of which 75% occur in adults; gliomata 15% of which 60% are in adults; gliosarcoma 5%, of which 80% are in adults. Tuberculosis, rarely primary, is usually secondary to some pre-existent lesion and basal in location. Syphilitic tumors, always acquired, constitute but 3.7%, 99% *of which are in adults.* These show a predilection for the *arteries, meninges and ocular nerves,* are usually small, may be multiple, and tend to extend to the adjacent cortex.* Cysts are ordinarily the result of hemorrhage or softening,

Statistical data.

* Starr states that gummata are not only rare in children but are never inherited and may appear within one year after infection or as late as twenty.—"Organic Nervous Diseases," 1903, p. 592.

rarely parastic. Gliomata and neurogliomata may be dense, but more often are soft or myxomatous and the seat of slight reactive inflammation or perhaps cystic changes; they occur most often in the substance of the cerebrum, cerebellum or brain stem. Osteomata, fibromata and lipomata are rare.*

Symptoms.—The most suggestive symptoms are: (a). *Persistent or paroxysmal, often violent, headache, usually at its maximum in the early morning.* (b). *Apparently causeless vomiting* most constant in children. (c). *Optic neuritis*, generally double. (d). *Mental changes* (usually apathy). (e). *Vertigo.* (f). *General or slight convulsions or epileptiform seizures* closely resembling either grand mal, petit mal, Jacksonian epilepsy, or even showing the psychic equivalents. (g). *Pressure symptoms*, irritative or destructive. (h). *Syncope.* (i). *Emaciation.* (j). *Polyuria.* (k). *Insomnia.*

Some of the more prominent symptoms.

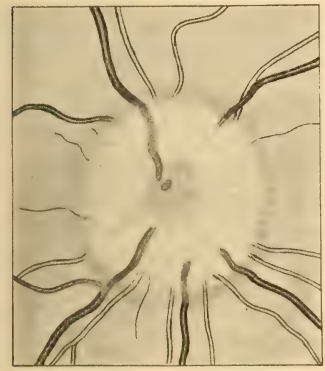


Fig. 228.—*Choked disc* cerebral tumor. (After Gowers.)

Large tumors in important locations may produce no characteristic localizing symptoms, or, their order and type may be suggestive or definitive. Unlike hemorrhage they are *usually* of *gradual onset* and *progressive in type* though inflammatory reaction may intensify them and periods of intermission are common.

Often symptom less.

Insidious and progressive.

GENERAL FACTORS IN RELATION TO TUMOR IDENTIFICATION.—Tuberculosis.—Three-fourths of all cases occur in persons *under twenty*, 50% *in those under ten years* of age. *A personal or family history of tuberculosis, predominance of basal lesion, or if cortical, irritative rather than destructive symptoms.* Retinal tubercle (late and rare). *Early progressive symptoms followed by prolonged intermission* and pontine or cerebellar localization.

The young chiefly affected.

Syphilis.—*History or symptoms of acquired, never a congenital*

*Starr's table shows that of 600 cases of brain tumor, 193 were tuberculous (152 in children), 120 were sarcomatous (86 in adults), 91 were gliomata (54 in adults), 30 were gliosarcomatous (25 in children), 32 were cysts (adults 30), 41 carcinomatous (31 adults), 22 were syphilitic (20 adults), 71 miscellaneous. Of all these the sarcomata most rapidly invade the adjacent tissues.

Always acquired.

syphilitic lesion, cortical localization and irritative lesions. *Rapid advance often followed by arrest*, headache worse at night. Disappearance under radical treatment, the effect of *antisyphilitic medication.**

Adults.

Almost exclusively a disease of adults.

Sudden onset.

Glioma.—There is usually *sudden coma* followed by accession of symptoms, irritative cortical phenomena.

Sarcoma.—Existence of a *primary growth, rapid, progressive and diffuse symptoms.* Common.

Carcinoma.—Elderly patients, usually over 50. Primary growth present. Local tenderness *may* be present in cortical tumors.

Terminal Symptoms of Tumor.—Rapid pulse, dementia, persistent continuous vomiting and Cheyne-Stokes respiration are sometimes present, stupor and coma common.

Focal Symptoms.†—*All lesions may involve other areas by contiguity, transference of irritation, actual pressure, or invasion.*

Cortex.—*Frontal.* Impairment of mentality, change of disposition, irritability, loss of memory, apathy and somnolence, childishness, fits of passion, maniacal outbursts or non-delusional dementia, motor aphasia and agraphia (left lower frontal). *Parietal.* Word blindness (left inferior parietal), hæmianopsia (if occipital lobe is affected), disturbed muscle sense and sensation opposite side of body, inco-ordination. *Occipital.* Mind blindness, *lateral homonymous* hemianopsia. *Temporal.* Intercortical sensory aphasia and word deafness (left side). *Sylvian Fissure and Island of Reil.* Pressure exerted by superimposed growths may involve internal capsule and cause *paraphasia* or hæmiplegia; inferior parietal or superior temporal, *sensory aphasia;* 3rd left inferior frontal, motor aphasia; operculum, facial palsy. *Motor* (Rolandic) *Area.* Jacksonian epilepsy (tonic or clonic convulsions) followed by transient, perhaps ultimately permanent paralysis and accompanied or preceded by localizing numbness or tingling (the origin and course of the spasm indicate the area of involvement); *spastic paralysis, i. e.*, increased reflexes, preserved muscle tonus, nutrition and electrical reaction.

Cerebellum.—*Median Lobe.* Optic neuritis and severe headache appear early, staggering gait or propulsive movements, vertigo (when upright) or actual falls. *Inferior Surface.* Associated cranial nerve involvement. *Middle Peduncles.* Unilateral swaying *in direction op*

* It should always be tried in doubtful lesions.

† Largely based upon the admirable descriptions of Starr, Nothnagle and Ziehen.

posite to growth, or propulsion and cranial nerve involvement *on same side* as lesion. *Superior Peduncles.* Same as corpora quadrigemina.

Corpora Quadrigemina.—Partial, usually unequal and bilateral oculo-motor palsy, nystagmus, staggering gait, superior and inferior recti chiefly affected, inco-ordination of eye movement, unsteady vision. These symptoms are pathognomonic.

Crus Cerebri.—Oculo-motor paralysis same side, hæmiplegia opposite side with perhaps bilateral hemianopsia (optic tract).

Pons.—Paralysis of 3rd and 5th nerves (upper pons), hence anæsthesia of face, *external strabismus*, corneal dryness and ulcer, dilated pupil, ptosis, etc. Knee jerks often inhibited. *Lower portion.* 6th, 7th and 8th nerve paralysis, *internal strabismus*, with alternating hæmiplegia. *Medulla.* Hemiplegia, hemianæsthesia, hemiataxia, paralysis of related cranial nerves including the hypoglossal, vagus and spinal accessory. The wealth of vulnerable structures in this region may yield a multitude of diverse symptoms. Ataxia, vertigo, projection movements, dysphagia, unilateral or bilateral sweating, vomiting, lingual paralysis, cervical retraction, etc.

Internal Capsule.—If this be affected there is hemianopsia, hemiplegia and hemianæsthesia. **Optic thalamus,** awkward positions of the body and athetoid movements, marked sensory disturbance and, in posterior lesions, homonymous hemianopsia; voluntary facial expression present, involuntary emotional expression absent. In most cases no characteristic symptoms.

CEREBRAL HEMORRHAGE, EMBOLISM AND THROMBOSIS.—Age.—Cerebral hemorrhage is rare before 40 and the tendency increases with age though cases are occasionally seen in children and in young men or women who have a precocious arterio-sclerosis. It is doubtful whether there is much difference in its incidence as between men and women. Hereditary predisposition certainly is of the highest importance (see page 47). Chronic alcoholism, lead poisoning and syphilis are important antecedent conditions as are gout and nephritis because of the associated high pressure and vascular changes. Traumatism and the hemorrhagic diseases such as hæmophilia, leukæmia and pernicious anæmia must be considered. The acute infectious diseases exert little influence. *Two factors are essential to* the ordinary type of cerebral hemorrhage, namely, *weakened vessels and high vascular tension. Hemorrhages may be large and single or punctate and multiple, cortical or "capsulo-ganglionic."* Arterio-sclerosis and atheroma of the senile type often associated with an hypertrophied left ventricle furnish most

Heredity, habits and occupation, and disease

Essential factors.

Miliary aneurisms.

of the cases, and one of the commonest of immediate pathologic causes is the rupture of such miliary aneurisms as may frequently be found in the medulla, pons, caudate and lenticular nuclei and optic thalamus, the lenticulo striate artery being the chief offender (50% of all cases).

Congenital or toxæmic.

Other cases may be due to congenital hypoplasia or acute toxæmic degeneration.

Absorption vs. Softening.

Absorption may take place in minor hemorrhages but ordinarily the brain tissue is destroyed by softening and the clinical symptoms depend both upon the position of the clot and its extent. Any hemorrhage that reaches the 4th ventricle will of course produce almost instant death. By extension from the neighborhood of the internal capsule exuded blood may reach and fill any of the ventricles. Cortical lesions result from similar lesions of the Sylvian artery and occasionally the *pons* or even the *cerebellum* may be the site of the rupture.

Hemorrhage into ventricles.

Exciting Causes.—Violent muscular strain, the sexual act, labor, defecation, fear, anger, joy and similar physical strains or emotional states most frequently excite an attack, but more often no definite cause can be assigned.

Cause evident or absent.

Premonitory Symptoms.—Usually none are present, occasionally *numbness, prickling* or *formication* in the lower extremities, *transient visual disturbance, irritability, headache, vertigo, drowsiness, or insomnia* may be noted before the attack.* Unilateral choreiform movements and spasm of the ocular muscles are rare, and in many instances such symptoms undoubtedly indicate a slight antecedent hemorrhage.† There are two distinct types of seizure, *the sudden* and *the gradual*, the former furnishing the typical cases of "*apoplectic insult*" or "*stroke.*"

Signal symptoms.

"Insult" or "stroke."

Symptoms of Attack.—*Loss of consciousness is immediate and complete, the face congested or ashen and cyanotic. All voluntary muscles are relaxed, the pupils may be moderately dilated, irregular, or, in ventricular or pontine hemorrhage, contracted. The pulse is slow, full and of high tension. There may be conjugate deviation* towards the lesion and the temperature is normal or more usually subnormal.

Striking signs.

Eye symptoms.

* In a case recently observed two attacks of momentary unconsciousness, mental confusion and sensory aphasia with barely perceptible transient tremor of the left hand were followed in a few days by an apoplectic seizure producing instant death. In another, on two occasions, the patient fell suddenly to the floor without stumbling or tripping, arose immediately, showing neither loss of consciousness, mental confusion or motor weakness, only to die instantly a few weeks later as in the preceding case. In one embolus seemed probable, in the other true apoplexy.

† The author has seen a number of cases in which ocular hemorrhages shortly preceded an apoplectic seizure.

Respirations are slow and stertorous, the cheeks are blown outward with each expiration, and *facial paralysis* may be indicated by the greater excursion of the paralyzed side. *The lips are relaxed, spluttering and foam covered.* In cases of gradual onset, paralysis and loss of consciousness may not appear for several hours. Larval cases.

Second Stage.—After a period varying from a few hours to one or two days there is a febrile inflammatory reaction lasting from one to 7 or 8 weeks. A preagonal hyperpyrexia or drop to subnormal usually indicates extending hemorrhage, rupture into the ventricle or involvement of the medulla or pons. After a variable period *consciousness, and usually to some extent motor power return* while in the limbs that remain paralyzed *early rigidity* develops. *Trophic changes* chiefly in the form of bed sores, and congestion of the lungs may add to the gravity of the case. *It is important that paralysis should be recognized in the cases of coma*, as it often at once differentiates apoplexy from other conditions. Ordinarily, it is only necessary to raise the arms and legs and note the comparative resistance offered to passive movement and the change in the tonus as indicated by the dropping of the lifted arm or leg. Facial paralysis is readily noted and eye symptoms such as conjugate deviation at once suggest the cause.* *Loss of power may be complete* and include the opposite half of the body including the area of the facial and hypoglossal nerve; *or, incomplete*, the face being unaffected. If the *ventricles* be affected *tonic rigidity or marked convulsive seizures* may occur. *The reflexes are entirely abolished during coma, though greatly increased when the first shock of the attack has passed*, and the theory of suspended function through shock seems a sufficiently reasonable explanation of this departure from the law that governs reflex action.

Reaction. Hyperpyrexia. Remission. Trophic changes. Signs of paralysis. Ventricular symptoms. Reflexes.

Residual Symptoms.—*The superficial reflexes* are absent permanently or for long periods over the affected side. *Conjugate deviation is usually transient* but occasionally persists for weeks or months and in these cases there is probably a direct lesion of the inferior parietal lobule. In *residual paralyses* we ordinarily find *crossed hemiplegia, usually most marked in the leg and least in the trunk with increased patellar reflexes. There is unilateral lagging of the thorax.* In respiration the shoulders drop downward, yet the muscles react normally to

Superficial reflexes. Paralyses.

*The most frequent source of hemorrhage is the lenticulo-striate region, a crossed hæmiplegia being produced rather by pressure than actual destruction which accounts for the marked diminution in extent and degree of paralysis so frequently following apoplectic insult.

the electric current and their atrophy is that of disuse. *Speech* is restored either entirely or to a considerable degree, often very early, but *dysphasia* may persist for some time. The well known *hemiplegic gait* is due to the patient's throwing the balance of the body constantly towards the sound side, *the affected leg swinging outward and striking the ground in a flail like manner.* Later flaccidity is replaced by spasticity, when, as a rule, predominating extensor contraction and a typical spastic gait appears (see page 35). Usually improvement in the leg is more rapid than in the arm and in the latter spastic contraction produces flexion of both elbows and fingers. A low grade arthritis from disuse is frequent with pain and partial anchylosis, symptoms of vaso-motor paralysis may appear and a secondary descending degeneration may intensify the deep reflexes even of the sound side. Hemianæsthesia affecting pain, temperature and touch is present though imperfect and ordinarily transient. *If marked and persistent* it constitutes a direct focal symptom. Muscle sense is less often affected but disturbances of vision and aphasia may be indirectly produced, and be transient or focal, persistent, or even permanent. Facial paralysis usually affects only the lower branches; the muscles of the neck and trunk are relatively less affected than those of the extremities and the hypoglossal paralysis is seldom complete. *Choreic or athetoid* movements may appear during the regenerative stage and usually indicates a focus in the posterior part of the internal capsule or the adjacent optic thalamus.

Aphasia and dysphasia.

Hemiplegic vs. Spastic gait.

Arthritis.

Secondary degeneration.

Hemianæsthesia.

Relative involvement.

Athetosis.

Localizing Symptoms.—*As regards the localizing symptoms it must be remembered that correct localization is seldom possible in the earlier stages of an apoplexy.* The persistence of a *hemianæsthesia suggests* a focal lesion in the posterior portion of the *internal capsule*, the *central convolutions or the intermediate medullary tissue. Monoplegia* indicates a lesion of the *centrum ovale, cortical motor centre*, or, a central paralysis of such nerves as the facial and hypoglossal if these are alone affected. If *aphasia complicate a monoplegia* a cortical lesion is most probable, *if aphasia be absent* the centrum ovale is suggested. *If hæmiplegia is associated with cranial nerve paralysis of the opposite side* from the 5th backward, a lesion of the pons or pons-medulla on the side of the cranial nerve paralysis is probable. If *ocular motor alternating paralysis co-exists with hemiplegia* a lesion of the peduncular tract is suggested. Hemorrhage into the internal capsule generally produces complete hæmiplegia.

Indefinite in early stage.

Important focal signs.

Differential Diagnosis.—*Symptomatic apoplexies*, that is those

pseudo-apoplectic seizures that occur in the course of such diseases as cerebral tumor or abscess and paralytic dementia may present the greatest diagnostic difficulty, though in most instances such a case history is known or obtainable as at once suggests their true nature. Absence of choked disc helps to exclude brain tumor. If hemianæsthesia appears it is usually transitory. Certain epileptic seizures resemble apoplexy but the subsequent course and previous history usually suffice.

Case history helps.

Condition of disc.

Uræmia.—An immediate diagnosis is often impossible even in the presence of urinary signs of renal disease. Convulsions are more likely to occur as an initial symptom of uræmia and the subsequent course is usually distinctive, uræmic paralyses being rare. *Meningitis.* This condition often offers serious difficulties particularly as certain apoplexies are associated with painful contraction and rigidity of the neck muscles. The persistence of convulsions, their bilateral incidence, the general hyperæsthesia, the possible demonstration of optic neuritis and the history and course of the case must be depended upon as indicating meningitis. *Acute sepsis* may produce pseudo-apoplexies but the clinical picture soon becomes distinct. *Hemorrhagic pachymeningitis* if unilateral cannot be differentiated from a cortical hemorrhage of slow onset.

Uræmic paralyses rare.

Hysteric Hemiplegia.—Hysteric paralyses are usually shifting and transitory and seldom involve the facial nerve or produce hemiopia. *Sensation is markedly* and usually atypically affected as compared with apoplexy. (See p. 615.) *There are usually visual, olfactory and auditory disturbances or hallucinations* and as a rule the well known *hysteric stigmata* make the diagnosis. As regards the secondary changes it cannot be decided positively by any known diagnostic test whether in a given case these are due to cerebral softening or to cerebral hemorrhage. Finally it cannot be too emphatically repeated *that focal disturbances cannot be correctly localized during the apoplectic seizure and judgment must in every case be suspended until the various and confusing indirect symptoms have had an opportunity to subside.*

THE DIFFERENTIAL DIAGNOSIS BETWEEN CEREBRAL HEMORRHAGE AND EMBOLISM.—*This depends almost entirely upon a consideration of the factors of antecedent disease and sometimes the age, as the symptoms of an actual attack are so nearly alike as to render positive differential diagnosis impossible in most cases.* Most important is *disease of the heart,* either rheumatic, arterio-sclerotic, myocardial, aneurismal or mere cardiac weakness.

Antecedent disease of the heart.

Associated conditions.

The attack may come on during quiet sleep, be transient and quickly recovered from and not infrequently there is a history of *antecedent embolism* in other portions of the body. *In certain cases a marked and sudden change in the character of the heart murmur may suggest the detachment of a particle of vegetation or auricular clot.* A large number of differential symptoms are given by different authorities, but neither the author's experience nor his knowledge of the conditions attending the two lesions lead him to give them any important place. *The significant fact that 90% of all cases of embolism depends upon an antecedent lesion of the left heart points the way to differential diagnosis.*

Heart murmur.

THROMBOSIS.—This lesion may be entirely symptomless even when not occurring in the so-called "silent" regions. The localizing symptoms and resulting paralysis are so nearly alike in thrombosis, embolism and hemorrhage as to permit no accurate differentiation from this standpoint. *The mode of onset of the paralysis in thrombosis is more suggestive,* the *symptoms being far less violent, coma seldom present* and *consciousness being as a rule either wholly or partially retained. Mental symptoms are much more pronounced than paralytic symptoms after the attack and usually assume the form of general mental impairment with marked irritability and a tendency to emotional excitement.* Finally the differential diagnosis of these diseases is assisted by the fact that *80% of the so-called apoplectic attacks are chargeable to cerebral hemorrhage,* and the physician's mind is also comforted by knowledge that an immediate *differential* diagnosis is of little importance to the patient.

Mode of onset.

Mental state.

Primary vs. Secondary.

CEREBRAL ABSCESS.—It might be thought that cerebral abscess would present striking general symptoms which differentiated it from cerebral tumors and other confusing lesions. *Such, however, is not the case save in exceptional cases, and the diagnosis ordinarily depends more upon the recognition of one of the well known causative factors than upon any specific or exclusive symptomatology.* Of such causes we may enumerate *traumatism with or without fracture, suppurative diseases of the scalp, empyema, pulmonary cavities and most important of all chronic suppurative otitis media,* a condition frequently associated with mastoiditis and caries. *Abscesses so caused are most frequently in the cerebellum,* whereas, when from other causes, they chiefly affect the cerebrum, abscesses of the basal ganglia or brain stem being rare. The accumulation may be superficial or within the substance of the brain, and 80% are single; multiple abscesses resulting usually from

Seek causative factors

remote foci uch as empyema. In size they vary usually from 2 cm. to 8 cm.

Symptoms.—*Extraordinarily latent cases have been reported which for several months or years yielded few or no indications of their presence. More often the symptom-complex is essentially that of brain tumor and consists largely of pressure phenomena.* In acute cases following operation or injury or in those secondary to an otitis media, the symptoms a e primarily those of a meningeal *inflammation*, and *there may be fever, chills, vomiting, delirium and severe headache*, perhaps preceded by symptoms that are irritative in character. In the latter stages of abscess, *mental torpor, drowsiness and optic neuritis* are common, *a period of latency* is the rule in cases which arise from distant foci and not uncommonly *from those due to direct injury.* The localizing symptoms are, of course, less distinct than in tumor but follow the same rules. *Fever is very often entirely absent, is often overlooked when present, if the primary disease be febrile and not infrequently there is subnormal temperature.* It is evident, therefore, that it is only by a combination of suggestive etiological factors, the symptoms of intracranial pressure and perhaps of septic absorption, that a differential diagnosis is made. MacEwen's method of skull percussion is more likely to yield results in abscess than in any other condition producing intracranial pressure. The *increased resonance* obtained depends upon a distension of the lateral ventricles and compression of the venæ galeni. *Optic neuritis* is far less constant than in brain tumor or meningitis.

Larval cases.

Brain tumor symptoms.

Meningeal form.

Fever.

Skull percussion.

Optic neuritis.

PARALYTIC DEMENTIA.—(General paralysis of the insane), (general paresis). **Etiology.**—The etiologic factors of general paresis are identical with those of locomotor ataxia and by the diseases seem to differ only in the seat of the morbid process, though alcoholic excess seems to be more important in the former than in the latter.

A disease of adult males.

Parasyphilitic.

Morbid Anatomy.—Amongst the multitude of diverse findings are: a small brain with shrunken convolutions, sclerotic arteries, hyperæmia, edema, hypertrophic pachymeningitis, thickened and adherent pia, an increase of cerebro-spinal fluid with dilatation of the 4th and lateral ventricles with granular ependyma. Microscopically there is general neuroglia overgrowth with degeneration of cells and fibre atrophy, and changes may sometimes be found in the spinal cord identical with those of locomotor ataxia. The whole brain is involved though in varying degree.

Symptoms.—As in locomotor ataxia we deal with a *parasyphilitic*

Syphilis chief factor

progressive degeneration ending in general exhaustion and palsy, but, unlike its congener, associated with marked mental symptoms and terminal dementia. It may be divided into two stages, to which some add a prodromal stage, and varies considerably in type.

Neurasthenic symptoms.

First Stage.—The early symptoms of this stage are significant but not distinctive, being identical with those of certain forms of neurasthenia. *A change of character* is soon evident, carefulness, good nature,

Change of character.

self restraint, frugality, good judgment, high ethical standards, concentration, vigor and modesty, give place to carelessness, in matters large and small, peevishness, irritability, or violent outbreaks of temper, periods of extravagance and foolish investment, coarseness and perhaps brazen immorality. The victim is inattentive to occupational and domestic duties and obligations, tires easily and lacks concentration and ability

Garrulity.

to perform sustained work. He is garrulous, egotistic yet jocose and

Delusions of grandeur

loud in voice and manner. *Delusions of grandeur* shortly appear and the patient deals only in superlatives. His enormous wealth, marvelously simple yet grandiose schemes, his house, his wife, his children,

Outbreaks.

his wonderful self are all beyond compare. *Violent outbreaks* may occur in this latter period of the first stage, often associated with drinking bouts and with sexual excesses often marked by extreme perversion.

Impaired memory.

In the later period of this stage or even during the earlier exaltation period lapses of memory may occur, *the handwriting may be uncertain, letters lack coherency*, errors in figuring occur and one notes *tremor*

Writing and speech.

of the facial muscles and tongue, difficult enunciation of consonantal words such as "British Constitution," the *speech being hesitant, slurring*

Knee jerks.

or even stuttering. The knee jerks are increased and the pupils often

Pupils.

sluggish and unequal or even of the Argyll-Robertson type. Attacks of *vertigo or syncope, pseudo-apoplexies with hemiplegia, or transient epileptiform seizures* may occur and *insomnia* may be troublesome and

Various symptoms.

intractable but *true apoplexy is uncommon* in this stage. *Sexual weakness* and *loss of vesical* power may be manifest but the vegetative function may be long preserved and the appetite is often gluttonous.

Second Stage.—*This is the stage of decided and increasing dementia, memory*, even for familiar faces and common events, becomes

Dementia.

fitful and almost lost. *Personal cleanliness* is neglected, there is *slobbering at meals* and a *gradually increasing dependency.* Apoplectiform attacks are followed by *permanent paralysis, hemiplegic in type, and true apoplexy may supervene and terminate the case. The duration* of this form is extremely variable, the stage of dementia being usually reached in a few months after the appearance of pronounced mental

symptoms. Death occurs sometimes within a year, but may be postponed for 15 or 20. *Remissions are not infrequent but entire recovery usually means an error in the diagnosis.* Duration. Remissions

Variation in Type.—*Dementia is sometimes primary, the period of exaltation and excitement being entirely lacking.* In such instances the premonitory symptoms *are almost precisely like those of pronounced neurasthenia,* in other cases a *marked hypochondriasis is substituted for exaltation.* In some of these cases there is much complaint of dull pain in the head, trunk and other extremities. *Other cases are of a primary exudative type* evidenced by primary ocular palsies or cephalalgia with convulsive seizures. *These cases are of especial interest* because of the fact that terminal symptoms of dementia are often postponed for five or ten years. Of equal importance are *those cases of taboparesis presenting a combination of locomotor ataxia and paretic dementia.* In these the onset may be primarily that of a locomotor ataxia, or, such as to immediately suggest the combined lesion. *When the cord symptoms are pronounced the brain symptoms are usually delayed and vice versa.* It must be remembered that *various forms of mental disease other than general paresis may complicate tabes dorsalis.* Primary dementia. Hypochondria. Cephalalgia. Primary cerebral palsies. Tabo-paresis. Cord vs. Brain symptoms.

Differential Diagnosis.—*The earliest stages of paralytic dementia in its ordinary form cannot be made the basis of an absolute diagnosis without risking a serious and dangerous error in diagnosis.* Practically every symptom may be found in curable cases of neurasthenia and in chronic alcoholism. *With the appearance of pupillary symptoms and marked tremor of the face and tongue, or marked and characteristic exaltation, the clinical picture is greatly cleared.* Furthermore, *cases of pure cerebral syphilis* exactly simulate one form of tabes and may lead to error. *A differential diagnosis may be impossible in such instances for a considerable length of time, pending the result of active and radical specific medication.* Early accurate diagnosis impossible. Cerebral syphilis.

Cyto-diagnosis.—In both tabes and paresis the spinal fluid contains a considerable amount of albumin and an excess of lymphocytes, but the value of the finding is limited to the proof that it offers of a subacute or chronic inflammation as contrasted with the polymorphonuclear leucocytosis of an acute inflammation. However, it fortunately occurs early in the disease, and may thus greatly assist the diagnosis. Early findings.

ANÆMIA OF THE CORD.—Only in the most profound acute and chronic anæmias do symptoms from the cord manifest themselves. In *pernicious anæmia* hemorrhage may occur or even a true postero-lateral sclerosis. Cord symptoms of severity may follow a great loss

of blood and on the other hand marked changes may be found at autopsy which have yielded no symptoms during life. The diagnosis can seldom be made save tentatively and is based upon the presence of anæmia as an etiological factor together with localizing symptoms.

THROMBOSIS, EMBOLISM, ENDARTERITIS OF THE CORD.—However important these conditions may be as the primary factor in certain degenerative changes they are by themselves of no clinical importance.

LOCOMOTOR ATAXIA.—(Tabes dorsalis), (posterior spinal sclerosis). **Definition.**—A disease characterized by a gradual onset and prolonged course, associated with loss of the deep reflexes and normal pupillary light reaction, visceral crises, inco-ordination, lightening pains and ultimate paralysis.

Adult males.

Parasyphilitic.

Etiology.—*It is essentially a disease of the male,* more common in the white than in the colored race, *occurs most frequently between the ages of* 30 *and* 50, *and is so generally associated with a past history of syphilis as to justify the belief that at least* 90% *of the cases are thus explained.* Alcoholic and sexual excess, exposure, fatigue and injuries are occasional associated conditions, but are probably not causative and usually not even contributory. Aside from hereditary syphilis which occasionally causes the disease in children, heredity plays no part.

Degeneration of sensory structures.

Morbid Anatomy and Pathology.—Much confusion exists and no definite statement as to the exact cause of the pathological changes is possible. *It is essentially a posterior spinal sclerosis* involving primarily the larger dorsal root fibres, though changes in the blood vessels of the nerve roots, pia mater and cord may appear early. The disease is a progressive degeneration and even though the primary lesion is not in the cord itself, the major changes are ultimately to be found there, and even macroscopic examination reveals the morbid condition in most instances.

Pupillary signs.

Dysuria and incontinence.

Knee and Achilles jerks.

Symptoms.—(Incipient stage). *Eye Symptoms. The Argyll-Robertson* pupil showing a loss of the iris reflex to light, with normal reaction to accommodation, is one of the earliest symptoms. *The pupils are often contracted,* perhaps unequally so, and there may be *optic atrophy, ptosis, paralysis of the external recti or even total blindness. Dysuria.* This is an early symptom, whereas incontinence occurs later in the disease. *Loss of Deep Reflexes.* Early in the disease the *knee jerk is diminished and finally lost,* the response being often unequal on the two sides until both are abolished, and the *Achilles jerk* may be lost first.

Pain.—*The characteristic lightening pains are acute, darting, stabbing, follow dorsal root areas,* are irregular in occurrence, variable in intensity, likely to be initiated by excesses of any kind and leave as suddenly as they appear. They most often affect the legs, but may occur in the arms, head or trunk, and sensitive areas and herpetic eruptions may follow them.

Often misinterpreted.

Ataxic Stage.—*Romberg's sign* is one of the earliest, the patient being unable to stand steadily with the feet together and the eyes closed. Furthermore, he cannot stand on one leg or start off promptly when ordered, his turns are made laboriously and cautiously and descending stairs is difficult. It may be present in the first stage and antedate the loss of knee jerks. *The characteristic gait* (ataxic) develops later, the patient throwing out the leg with jerky, uncertain action, keeping the feet far apart to secure a wider base, and bringing them down heel first with a sort of stamp; being obliged ultimately to use one or even two canes or totally disabled. *Inco-ordination is less prominent in the arm movements than in those of the leg* and muscle *power* is remarkably preserved, as is nutrition, until the later stage of the disease. There is *unusual mobility and relaxation of the joints* which occasionally appears at a comparatively early stage. *Various sensory symptoms* develop during the ataxic period, most marked in the lower extremities, the patient feeling often as if the feet were muffled in cloths or cotton and losing the sense of the resistance of the ground. Sensations of numbness and tingling are prominent and lightening pains may or may not exist in varying degrees of severity. Not only is the *tactile sense* impaired but the *sense of pain* as well, and this is associated with a marked retardation of pain transmission so that several seconds may elapse before the prick of a pin is perceived. *Oddly enough ataxic symptoms are unusual or at least greatly delayed in those cases in which optic atrophy is an early sign* (*one in ten*).

Static ataxia.

Ataxic gait.

Inco-ordination.

Muscular power.

Joints.

Anæsthesia

Paræsthesia.

Analgesia.

Significance of optic atrophy.

Visceral Crises.—*These remarkable symptoms constitute a frequent source of error in diagnosis and may be gastric, intestinal, rectal, renal, urethral, clitoral or laryngeal and associated with marked pain in and functional disturbances of the parts affected.* At this stage incontinence of urine may displace simple dysuria or retardation, cystitis and even pyelo-nephritis may occur; sexual power, diminished in the early stage, is now usually lost, and simple constipation may be replaced by *critical rectal pain and spasm and ultimately by relaxation of the sphincter.*

Misleading.

Urinary and rectal symptoms.

Trophic Changes.—*Perforating ulcer of the foot and the so-called Charcot's joint* are the most prominent of these changes. The former

Charcot's joint and perforating ulcer.

appears usually on the sole of the foot beneath the great toe, the latter is a *painless disintegration of the joint associated with effusion, deformity and tendency to dislocation.* The bones are brittle in this disease and spontaneous fracture may occur. Various skin rashes, areas of sweating, herpes and edema may appear in connection with lightning pains. Muscular atrophy seldom appears until late in the disease but the nutrition of the nails may be greatly affected. *Brain Symptoms.* Paranoia, melancholia, and dementia may occur, and paralytic dementia *is* "cerebral tabes," the difference being merely one of localization. *Duration.* Paralyses are seldom marked until from 5 to 8 years have elapsed; even then progress is slow, periods of apparent arrest not uncommon, while actual arrest sometimes occurs. The chronic course of the ailment is remarkable, the patient may live for 25 or 30 years, though rarely cases occur in which the progress of the disease is rapid. Death results from *intercurrent disease* in nearly every instance.

Cutaneous symptoms.

Loss of power delayed.

Long duration.

Differential Diagnosis.—Tabes need seldom be confounded with any other disease, *the combination of loss of deep reflexes, the Argyll-Robertson pupil* and lightening pains making a distinct clinical picture. In true ataxic paraplegia the knee jerks are increased, in chronic multiple neuritis with ataxia ("pseudo-tabes") there is marked muscular atrophy and tenderness over the affected nerves.

ATAXIC PARAPLEGIA.—(Gower's disease, postero-lateral sclerosis, etc.). A combination of *marked inco-ordination and slowly developed spastic paraplegia* with *retained and increased reflexes and frequently ankle clonus makes a clean cut clinical picture.* It is associated with degenerative changes in the dorsal and lateral columns of the cord and is of unknown causation. The sphincters are usually involved, there is a late development of mental symptoms similar to those of paretic dementia, and cranial nerve symptoms are seldom pronounced. There may be "dull aches," rarely lightning pains.

Typical cases simple.

PRIMARY COMBINED SCLEROSIS.—This name has been given by Putnam to cases presenting clinically *progressive weakness, numbness of the extremities, exaggerated tendon reflex, spastic contraction and paraplegia* with *pronounced anæmia.*

HEREDITARY ATAXIA.—(Friedreich's ataxia). This disease of childhood or young adults needs another name as it is not invariably hereditary. It is associated with degeneration of the dorsal and lateral columns of the cord.

Also postero-lateral.

Symptoms.—The chief features are *marked inco-ordination involving first the legs, but later the arms, and these to a marked degree.* The *gait*

is uncertain and staggering rather than stamping. Arm movements are jerky and irregular, grasping involves pouncing upon the object. Tendon reflexes are diminished or lost, there is early talipes equinus, the big toe becomes flexed dorsally, nystagmus occurs. Pupillary reactions and the optic nerve are *unaffected. Paralysis and mental impairment* usually come on late. The speech is indistinct, slow and scanning.

CEREBELLAR HEREDITARY ATAXIA.—(Marie). This *later life* form of hereditary ataxia, presumed to be associated with a congenital cerebellar defect, is characterized by *impaired pupillary reaction, exaggerated tendon reflex, clonus, ocular palsies, optic atrophy, and, its development in the third decade. Inco-ordination, nystagmus and scanning speech are present as in Friedreich's ataxia, but the gait is more distinctly reeling and drunken.* It is doubtful if any line should be drawn, as in the two diseases the same general tracts are affected, the spine in Friedreich's ataxia, the cerebellum in that of Marie, the actual tracts being structurally continuous.

PRIMARY LATERAL SCLEROSIS.—(Spastic paralysis of the adult). This is a disease of early adult life, most common between the ages of 20 and 40, no definite etiologic factors have been proven and the pathologic change is essentially a degeneration of the pyramidal tracts, either primary or secondary.

Symptoms.—*Muscular rigidity and weakness* are often associated with *dull pain* and a *persistent sensation of fatigue and rigidity* and *impairment of locomotion slowly increase and are out of proportion to the loss of power. The spastic gait is marked early in the disease and later becomes extreme,* the patient *shuffling* along with toes dragging, *tripping over every obstacle, the legs being closely approximated* or even so crossed that locomotion is lost. The *knee jerk is extremely active,* Babinsky's sign, *rectus and ankle clonus* are easily elicited, *superficial reflexes are increased* and in some instances clonus appears whenever the foot is placed upon the ground and the slightest touch may produce *clonic spasm* of the legs. The latter may be equally or unequally affected and the arms are usually free until the disease is far advanced. The ailment is one of long duration, yet nutrition is remarkably preserved, sensation is not affected and the sphincters are rarely involved until late in the disease. Ultimately an extreme state of disabling contracture and atrophy from disuse develops. Diagnosis is never positive, as primary sclerosis is excessively rare and cases usually prove to be secondary.

Spasm and lost power.

Spastic gait.

Increased reflexes.

Atrophy terminal only.

Secondary Spastic Paralysis.—It is hardly possible to separate

secondary from primary spastic paralysis which may result from any condition producing a lesion in the pyramidal tract, i. e., compression, tumor, caries, etc.

HEREDITARY SPASTIC SPINAL PARALYSIS.—(Hereditary spastic paraplegia), (family form of spastic spinal paralysis). *This variety is distinguished from infantile spastic paralysis chiefly by its distinctly hereditary nature and the fact that it commences several years after birth,* usually at about the age of 5. *Two groups* are distinguished, viz.:—those with the symptomatology of a cerebral *spastic paraplegia* developed in infancy and childhood, but *lacking all other cerebral symptoms*, and those developing in early adult life insidiously with primary spasticity and late paralyses.

Heredity and age important factors.

THE SPASTIC PARALYSIS OF INFANTS.—(Birth palsy), (spastic cerebral paraplegia), (Little's disease), (spastic diplegia). This condition is essentially a *general spastic paralysis occurring at or shortly after the time of birth,* more rarely following a convulsive seizure or an acute disease. *The legs* are chiefly affected without marked *sensory disturbances or wasting and the reflexes are increased. Athetosis and ataxia are extreme and the mental condition markedly affected.* The condition is no doubt most often due to meningeal hemorrhage and the interference with mentality will depend upon the extent and location of the effusion. In cases of premature birth spastic paraplegia without mental symptoms is thought by some to be due to an imperfect development of the pyramidal tracts. *In most instances, however, the disease is associated with instrumental delivery and seems to be due to actual injury.*

Spastic type.

Infancy.

Normal sensation.

Increased reflexes.

No atrophy

No R. D.

Backwardness of the child may first call attention to the condition, or there may have been *repeated convulsive seizures. The head may seem badly supported, the child may not be able to sit up and shows no tendency to walk or creep at the proper age. Leg rigidity and adductor spasm* are usually marked. **Symptoms** *are commonly either absent or less marked in the arms;* when marked it *constitutes* a *spastic diplegia. Constant irregular larger movements* of a choreic type may be present in the extremities, sometimes most marked when co-ordinate movement is attempted and pronounced bilateral athetosis is often present.

Backwardness and mental defects.

Rigidity and spasm.

Oscillation.

Athetosis.

Erb's Syphilitic Spinal Paralysis.—The leading features are *slight* muscular rigidity, *exaggerated deep reflexes, pain and sensory disturbance trifling, involvement of the rectum and bladder, impotence, a slow onset and a certain amenability to treatment.*

Slight rigidity.

Involvement of sphincters.

HYSTERICAL SPASTIC PARAPLEGIA.—Hysterical persons can accurately simulate true spastic paraplegia and differentiation is sometimes extremely difficult, but a few positive points can be laid down. Such are: *spurious clonus, more marked and irregular disturbances of sensation, variability in the site and intensity of paralysis, and unsustained or irregular resistance to passive movements*, and the frequent association of hysterical stigmata such as aphonia, meteorism, and excessive tenderness in hysterogenic zones.

Spurious clonus.

Peculiar resistance.

AMAUROTIC FAMILY IDIOCY.—The chief characteristic of this form of infantile paralysis affecting children during the first or second year of life is the constancy of blindness, first, partial, later, complete, associated with optic atrophy. The children become idiots and either spastic or flaccid paralysis may develop. It is a family disease, kills usually before the end of the second year, and is sometimes associated with auditory disturbances, strabismus and nystagmus.

Blindness and idiocy.

Death.

Fig. 229.—Anæsthetic area common in hysterical paraplegia. The genitals are spared. (After M. Allen Starr.)

SYRINGOMYELIA.—Pathologically, this disease consists in the formation of embryonal neuroglia tissue which undergoes hemorrhagic or degenerative changes, resulting in an enlargement of the central canal of the spinal cord quite distinct from ordinary dilatation; it is a disease of early adult life, almost invariably involves the cervical region, sometimes assumes a family type and affects men much more frequently than women. *A most pronounced diagnostic feature is a loss of thermal and painful sensation without marked involvement of the tactile or muscle sense.* This is usually accompanied by *pain in the extremities and cervical region, followed by peripheral muscular atrophy of the hands, slowly extending centrally. Scoliosis is usually present, the legs may*, late in the disease, *present spastic paraplegia* and *cutaneous disturbances both trophic and vaso-motor are common.* Such are dry or sweating skin, congestion or edema of the extremities, especially the hands, bullous or herpetic eruptions, atrophy and disintegration of the nails, erosions, fissures and ulceration of the terminal phalanges. *It should be noted that these cutaneous lesions are painless.* Spontaneous fractures and arthropathies may occur and late in the disease the vesical, rectal and genital centres may be involved and bulbar symptoms appear. Reflexes are usually increased and fre-

Cervical region chiefly.

Dissociated anæsthesia.

Spinal symptoms.

Painless cutaneous lesions.

quently the general aspect is that of amyotrophic lateral sclerosis, though if the dorsal columns are involved, tabetic symptoms may be marked. Clinically, one may encounter a *straightforward type* lacking the predominance of confusing motor or sensory changes, *or the picture may be that of amyotrophic or spastic paralysis, tabes, hysterical hemiplegia or a type in which trophic changes predominate.*

Mixed types.

MYELITIS.—May be acute, *subacute or chronic* and the term is usually confined to a lesion involving both gray and white substance, *poliomyelitis and focal* (involving a limited area) *myelitis* being separately considered. As regards the position and extent of the lesion we distinguish cases that involve a considerable vertical area as "diffuse," those affecting whole transverse area as "transverse" and those with or due to crushing or direct traumatic solution of continuity *disseminated* foci.

Varieties.

COMPRESSION MYELITIS.—This type is associated with traumatism, spinal caries, aneurism, new growths and parasites, more rarely with large syphilitic growths.

Diagnosis.—The diagnosis depends somewhat upon the recognition of an *antecedent caries, aneurism, malignant growth or syphilitic injection.* This form of myelitis is as a rule *much more painful than acute myelitis* and in malignant disease especially most excruciating suffering may be witnessed from compression of the nerve roots, either directly by the bones, a tumor, or an exudate. *In unilateral compression* the Brown-Sequard's syndrome (p. 526) may be evident. *Paraplegia* in these cases is usually slow in development and preceded by weakness and paræsthesia, the girdle sensation is usually marked and sensation persists longer than motion. If the cord be involved above the lumbar region, the paralysis is spastic with exaggerated reflexes; if below, the reflexes are usually diminished or lost and the sphincters involved. *Recovery may be complete* in cases of pressure due to deformity if the latter be corrected.

Seek cause.

Note pain.

Brown-Sequard type.

ACUTE MYELITIS.—*An inflammation of the cord substance may follow or more rarely directly complicate acute infectious diseases*, chief of which are variola, severe dysentery and acute intestinal toxæmia, diphtheria, typhus, acute rheumatism, measles, septicæmia and pyæmia, gummatous or endarteritic syphilis. Less important are erysipelas, pneumonia, malaria, puerperal fever, scarlatina and influenza and certain acute chronic intoxications such as alcoholic and gas poisoning and gout. Traumatism, with or without frac-

Causes.

tures (sometimes mere concussion, violent torsion or flexion), caries, and not infrequently a combination of fatigue and exposure to wet and cold may initiate an attack. It may also be due to the extension of meningitis or even neuritis, or develop secondarily in tuberculosis and syphilitic processes and *it occurs chiefly in men* and *between the ages of 20 and 40.*

Sex and age.

Symptoms.—*Premonitory malaise, sense of weakness and numbness, chill or chilliness and a variable degree of fever accompany the attack.* One must *first diagnosticate a myelitis, secondly, determine the position and extent of the lesion; and finally, determine the probable cause as a factor in prognosis.* The essential symptoms are, (a). *Hyperæsthesia, absence of reflexes* and *a girdle pain* (painful constriction) *at the level of the lesion.* (b). *Rapidly developing, not immediate, motor paralysis* below the lesion chiefly affecting the flexors level, partial or complete, according to the degree of transverse involvement. (c). *Increased reflexes* below the lesion. (d). *Involvement of the sphincters,* rectal and vesical. (e). *Vaso-motor phenomena,* i.e. congestion, sweating. (f). *Trophic changes,* especially pressure necrosis over the heel, hips and other dependent portions. (g). *Changes in muscle tonus, nutrition and electrical response varying with the level of the lesion.* The actual level of the lesion is best indicated by the upper margin of anæsthesia, the hyperæsthetic level being just above it. It will be seen at once that the region involved markedly affects the motor symptoms. If in the *lumbar enlargement the paralysis is typically flaccid,* with atrophy, loss of knee jerks, R.D. and urinary incontinence. *If mid-dorsal or cervical* the paralysis is *spastic and the reflexes are usually lost only in the areas corresponding to the actual level of the lesion. The picture being ordinarily that of spastic paraplegia with retention of urine, increased muscle tonus, Babinski's sign, ankle clonus and patellar reflex.* In the *cervical form, flaccid paralysis* may be marked and relatively extensive at the level of cord involvement *and if the 4th cervical roots be involved death may result from paralysis of the diaphragm, and vomiting, bradycardia, syncope, hiccough and dyspnœa may occur. Disseminated foci* must be recognized by regional diagnosis. *The upper level of a transverse or diffuse lesion is clearly indicated by the girdle pain and zone of hyperæsthesia, which may be emphasized by passing a hot cloth or sponge over the spine. Optic neuritis and myosis* may occur in cervical myelitis. Symptoms of irritation (spasm or convulsions, pain) if present are usually of short duration, but the diseased area tends to extend. *If diffuse there is acute ascending paralysis.*

Premonitory.

Localizing.

Ordinary type.

Lumbar type.

Mid-dorsal.

Flaccid paralysis at lesion level.

Upper boundary zone.

Unusual signs.

Differential Diagnosis.—*Hemorrhage* into the cord is characterized by absolute suddenness of onset, and frequently lacks fever. *Abscess, by suppurative foci;* in *meningitis* the irritative, painful and spasmodic symptoms predominate, increasing with disease duration until stupor or coma supervenes, whereas in *myelitis* they are relatively slight or of brief duration. The diseases may of course co-exist. Only a dorsal myelitis could be confounded with hysteria, and here girdle sensation, sphincteric involvement, true ankle clonus with perhaps extension spasm and the trophic changes are distinctive. In *Landry's paralysis* the lesion is an *ascending motor paralysis, flaccid throughout,* with normal electrical reactions, lost reflexes, and usually, good sphincteric control. *Multiple neuritis* presents at times great difficulty, but ordinarily the *more gradual onset* of paralysis, *marked pain and local tenderness over peripheral nerve areas,* and the *less frequent sphincteric disturbance* makes the diagnosis clear. The combined cases offer more difficulty. *Acute Anterior Poliomyelitis.* This shows flaccid paralysis throughout, seldom involves the sphincters, and lacks the gi dle pain and other sensory and trophic phenomena of myelitis. *Subacute Myelitis.* Differs from the acute only in its gradual onset (3–6 weeks).

Hemorrhage and meningitis.

Hysteria.

Landry's paralysis.

Neuritis.

Poliomyelitis.

Subacute and chronic myelitis.

Primary Chronic Myelitis. The only essential factors are its slow and irregular development, affecting at random the different cord functions, motor, sensory and trophic, but lacking the nerve root symptoms of compression myelitis. *Compression Myelitis.* Is suggested by the history or presence of spinal caries, traumatism or new growths, aneurisms, syphilis, echinococcus or cysticercus cysts or syphilitic infection, combined with the symptoms of a slowly developing myelitis, persistent and perhaps agonizing pain if the nerve roots be involved (malignant growths and metastatic cancer causing the most severe type), and *such pain may be marked over areas anæsthetic to painful and thermic stimuli* (anæsthesia dolorosa). The relation of pain *to spinal flexion may be suggestive, as is its relief in properly treated cases of caries and deformity.* Aside from the areas directly involved spastic motor paralysis predominates, save in the lumbar region, and sensory paralysis varies with the transverse area of the lesion. The Brown-Sequard syndrome may of course be present, indicating unilateral involvement. This symptom complex is more common here than in any other spinal cord lesion save hæmatomyelia.

Compression myelitis.

Anæsthesia dolorosa.

Brown-Sequard syndrome common.

Course.—As any region, grade or type may be involved, the course varies from early death to complete recovery or the establishment of chronic myelitis and permanent deformities. *Sensation,* last to go,

is first to return. In unfavorable cases *bed sores* may hasten the end, sometimes being associated with *hyperpyrexia;* descending and ascending sclerosis may occur; the legs may become rigid or the seat of flexor or extensor spasm (i.e. "*jackknife rigidity*"), and the *reflex responses extreme. Urinary decomposition* may produce *cystitis* or *pyelitis* and hasten the end. *It should be remembered that cases of slow onset and a history of antecedent acute infectious disease yield the best prognosis. Bed sores, high degrees of anæsthesia, sudden severe onset and an unusual degree of early pain and spasm are bad omens.* Good habits, relative youth, a vigorous physique, and an absence of tuberculous or malignant disease are encouraging. In *chronic cases after two years no hope of recovery may be entertained.*

Determining factors.

Chronic.

LANDRY'S PARALYSIS.—This rare disease of unknown causation chiefly affects *young adult males* and is characterized by a *progressive ascending paralysis,* and a sudden onset suggesting an acute infection or toxæmia. **Symptoms.**—*A sharply febrile onset is succeeded by a complete paralysis beginning in the legs and involving in a few days or even hours the trunk, upper extremity, muscles of respiration and usually of deglutition and articulation.* Enlargement of the spleen may be noted, *the sphincters are usually not involved, the reflexes are lost, but sensation is ordinarily preserved, or but slightly affected and the muscles do not atrophy or show abnormal electrical reactions unless ailment is of unusually long duration.* **Prognosis.**—Death in from 48 hours to two weeks is the almost invariable result.* Nothing definite can be said of the pathologic changes save that the lower motor *neurones* are the point of attack. *Acute myelitis* shows anæsthesia, bed sores, reaction of degeneration and atrophy at the level of the lesion and early *involvement of sphincters. Multiple neuritis* cannot be positively differentiated in certain rare instances (see multiple neuritis).

Rarely descending *not* afebrile.

Sensation preserved.

Sphincters rarely involved.

Atrophy and R. D. usually absent.

POLIOMYELITIS ANTERIOR ACUTA.—(Acute anterior poliomyelitis). **Etiology and Morbid Anatomy.**—This acute and destructive disease is probably due to primary thrombus or embolus involving the arterial supply and hence the nutrition of *the motor cells of the anterior horns, with occasional slight meningeal involvement; hemorrhagic myelitis followed by atrophy being the essential change.* In long standing cases the dependent anterior nerve roots degenerate, and atrophy of the muscles and the reaction of degeneration follow in a few

*The author kept a young girl alive for 41 days by artificial respiration, breathing having ceased early in the attack. Muscle atrophy was well marked in later stages.

Children chiefly.

Occasionally epidemic.

days the sudden onset of paralysis. *Children under three years of age are chiefly affected,* sometimes when in perfect health or, after some infectious fever, and occasionally it assumes an epidemic form. Adults are seldom attacked, but if so the symptoms are identical with those to be described.

Symptomatology.—The onset is variable; in certain cases the paralysis is unconnected with any definite febrile attack; more commonly there is slight transient fever and malaise, while rarely, high fever, diarrhœa, vomiting, delirium, convulsions and coma may be present. The more severe the primary attack the more rapid is the onset of paralysis.

Variable onset.

Pain and tenderness in the affected limbs may first attract attention but are usually absent or unimportant. The paralysis may involve all four extremities, rarely one, more usually both legs and especially the peroneal and tibial groups, and tends to recede in certain muscle groups and become marked and permanent in others. Whenever a large muscle tract is involved great inequality of damage is evident and certain muscles such as the forearm extensors are almost immune.

Flaccid paralysis.

Distribution.

Atrophy, lost knee jerks and R. D.

Secondary contractures occur late in the disease. It should be remembered that *the onset of this disease is sudden, the paralysis appears within from 24 to 48 hours, the lesions soon become fixed, the paralysis is flaccid, atrophy rapid and extreme, reflexes and normal electrical reactions lost, sensation unaffected,* and, that the sphincters and facial muscles are rarely involved.

Prognosis.—Seldom fatal, the disease is nevertheless a terrible one by reason of the persistence of the paralysis. In certain cases, due evidently to toxæmia, brisk and persistent eliminative treatment may result in complete or partial recovery. Rarely a recoverable case of neuritis may present almost identical symptoms.

Usually permanent disability.

PROGRESSIVE MUSCULAR ATROPHY.—(Amyotrophic lateral sclerosis), (chronic anterior poliomyelitis), (progressive bulbar paralysis). This is a disease of unknown causation, affecting chiefly adult males beyond the age of 30, said to be influenced etiologically by the usual factors, such as traumatism, excessive mental or physical strain, exposure to wet or to cold, and, to a slight degree, hereditary influence. *It is essentially a gradual degeneration of the motor neurones, either lower or upper being first involved, the lower in the greater degree.* The degenerative process affects the gray matter of the anterior horns and fibres of the anterior nerve roots within and without the cord, and, the intermuscular branches. The degeneration of the gray matter extends to the medulla and the antero-lateral white tracts and lateral

Adult males.

Usual factors.

Motor neurone degeneration.

pyramidal tracts are affected in varying degree, in some cases even to the motor cortex. There is of course marked muscular atrophy.

Symptoms of the Aran-Duchenne Type.—*Following a period of vague and indefinite pseudo-rheumatic pain, muscular atrophies appear in the following order. The muscles of the ball of the thumb* (the patient first noticing an inability to adduct the thumb, separate the index from the middle finger, write, or button his clothes), *the interossei and lumbricales, forearm flexors* and *extensors and* the *deltoid*, followed by other muscles of the shoulder girdle group. One hand is usually first affected and the muscles of the legs and face and the trapezius are usually attacked late. The platysma myoides *entirely escapes and indeed may hypertrophy* and *fibrillary contraction* is marked. The atrophy of the muscles of the hand and forearm produces the so-called "*claw hand*" and the wasting of intercostal and abdominal muscles reduces the patient to a helpless skeleton. Later the neck and even in some instances those of the face are involved and in prolonged cases those of the legs. The electric excitability of the muscles is early diminished and finally lost, the reaction of degeneration occurring in rapidly advancing cases. *Muscular power of course disappears pari passu* with the wasting. *Subjective sensory symptoms* may be present yet sensation is unimpaired and the sphincters unaffected, but the knee jerks are lost *when the quadriceps extensor atrophies.*

Order of involvement.

Fibrillary contractions.

Trapezius late.

Platysma not at all.

Knee jerks long retained.

Sensation unaffected.

Amyotrophic Lateral Sclerosis.—In certain cases of a *progressive muscular atrophy* described as *amyotrophic lateral sclerosis* a primarily spastic paralysis is associated with atrophy following the Aran-Duchenne order quite closely. In these the reflexes are increased and the picture is one of spastic paraplegia with the typical gait but with gradual atrophy. *Jaw clonus* and Babinski's sign are present but the sphincters are unaffected. The tendon reflexes are seldom increased in the flaccid type, markedly increased in the amyotrophic type. *Fibrillary contraction* is marked and there is a generally exaggerated motor response to muscle or tendon percussion, yet there is usually a partial reaction of degeneration. Bulbar involvement is often early and may precede the leg involvement.

A blend of anterior poliomyelitis, spastic paralysis and bulbar palsy.

The Bulbar Type.—(Glosso-labio-laryngeal paralysis). *Bulbar symptoms may precede or follow spinal symptoms but no actual pathological distinction exists to justify making of the primary bulbar type a separate disease.* Bulbar involvement is indicated by *difficulty in the pronunciation of the linguals and dentals, atrophy and weakness of the tongue leading to dysphagia, dysarthyria,* the accumulation of saliva

Fibrillary contraction.

and drooling. With involvement of the lips, labial pronunciation becomes difficult and succeeding paralyses of the pharyngeal and laryngeal muscles increase the dysphagia and enfeeble the voice. This type may be associated with progressive atrophy of either the tonic or flaccid type and cases of acute onset occasionally occur. A similar condition affecting the eye muscles is known as *progressive ophthalmoplegia.*

Acute bulbar form.

Muscular Atrophy of the Peroneal Type.—This differs from the preceding form in three particulars, *first, that it is an hereditary or family disease. Second, in that the atrophy commences in the muscles of the feet and peroneal group*, often unilaterally at first, this being the reverse of the usual form. *Third.* Sensory disturbances are prominent; it especially affects young children, and leads to various forms of club foot and the sphincters are not involved.

Peroneal ascending form.

Fibrillary contraction.

Differential Diagnosis.—The chief difficulty arises in distinguishing between a progressive muscular atrophy of the spastic type and syringomyelia, differentiation being only possible when the peculiar retained general sensation with sensory symptoms of the latter disease are pronounced. The bulbar type must be distinguished from the *pseudo-bulbar palsy* of cortical origin which results from bilateral lesions in the facial area of the cortex or between these areas and the cranial nuclei, *which lacks the fibrillary tremor, rapidly advancing atrophy and R. D. of bulbar palsy.*

MUSCULAR DYSTROPHIES.—There are various clinical forms of muscular wasting, with or without initial hypertrophy, classed under diseases of the nervous system but lacking definite pathologic findings. *Atrophy, palsy and marked hereditary tendency usually co-exist in all forms.*

No pathology.

Etiology.—The disease may exist in a family for generations and is most frequent in the male. Atavism similar to that of hæmophilia is evident, the mother transmitting the disease even though herself unaffected.

Heredity.

Classification.—No sharp dividing line can be drawn, but for clinical purposes cases may be divided into (a) *those occurring in youths and adults*, (b) *those of childhood.* Class (a). The disease may assume one, *atrophic* and two, *hypertrophic* forms. The former including (a) *cases without involvement of the facial muscles*, (b) *those in which they are early involved.* The latter includes both cases of genuine hypertrophy, and those of muscle lipomatosis.

Two classes.

PSEUDO-MUSCULAR HYPERTROPHY.—Symptoms.—*Motor impairment calls attention to apparent enlargement of the muscles* usu-

ally first of the calves. The leg extensors, glutei, the lumbar muscles, the deltoid, the triceps and infraspinatus are excessively involved and become prominent and resistant yet *weakness co-exists and is progressive.* Placed upon the floor the patient passes through a succession of characteristic movements to regain a correct posture. (See fig. 230.) When standing there is *lordosis, the shoulders are thrown back, belly prominent and feet widely separated. The shoulder girdle being greatly weakened and extremely movable makes the arms abnormally* long *and yields a sensation of slipping if an attempt is made to raise the child by catching him under the armpits.* The terminal picture is that of *extreme atrophy and helplessness* without fibrillary twitching or R.D. but *associated with mental degeneration.*

Prominent weak muscles.

Posture.

A lifting test.

Terminal stage.

Fig. 230.—Characteristic postural changes in pseudo-hypertrophic paralysis accompanying act of rising from floor. (After Gowers and Starr.)

Duration of the disease is from a year to two decades or more, death occurring from intercurrent disease or inanition, and prolonged remissions may occur.

Differential Diagnosis of Muscular Atrophies.—The chief difficulty in diagnosis arises in differentiating between certain cases of the disease which present overlapping symptoms, it being sometimes impossible to assign a case definitely to a single division. It should be remembered that in the *pseudo-muscular hypertrophy* the typical symptoms as just described are present, whereas in the *facial type the first changes appear in the muscles of the face, particularly the orbicularis oris and extend to the muscles of the cheeks and forehead, affecting both expression and speech and producing what is known as the tapir mouth. The extension to the general muscular system is downward. Erb's juvenile muscular dystrophy* is characterized by its being *distinctly a disease of youth, having its incidence usually at the age of puberty and within the age limit of* 12 and 20 *years* (12–16 usually). Furthermore that *the muscles first involved are those of the shoulder girdle. In all forms* late contractures are of course common with the usual deformities. *Fibrillary contraction of muscle, reaction* of *degener-*

Facial type.

Erb's type.

ation, disturbance of sensation and reflexes are lacking until extreme atrophy and weakness have developed.

DISEASES OF THE MUSCLES.

MYOTONIA.—(Thomsen's disease). This is a disease of childhood, of the family and hereditary type, affecting chiefly the male, and very rare in this country. Occasionally cases of the acquired type may be encountered.

Symptoms.—The characteristic symptom is a peculiar slowness of muscular contraction and relaxation, most marked in prehension and locomotion. The hand closes slowly and awkwardly around the object, and relaxation is equally deliberate. So in walking the leg is slowly advanced and halts for a second or two, yet the awkward gait lasts for but a few steps. The laryngeal, ocular and facial muscles are rarely affected, the general musculature is well or unusually developed and out of proportion to the actual strength. As might be expected the electrical reaction of the involved muscles is deliberate and associated with vermicular contractions between the poles. The disease is essentially chronic and incurable.

Peculiar complex.

Erb's myotonic reaction.

MYÆSTHENIA GRAVIS.—(Asthenic bulbar paralysis. Erb-Goldflam's syndrome). This is a progressive loss of muscular power associated with rapid exhaustion under faradic stimulation though not under galvanic (Jolly's myæsthenic reaction). The loss of power affects first the muscles of the eye, face, jaws and neck, but may involve the entire body. The essential symptom is the rapid fatigue of the affected muscles, restored by rest, and this in connection with the peculiar electric reaction, the transient nature of the symptoms, simulating paralysis, and the absence of true atrophy or fibrillary contractions makes the diagnosis easy. The disease is essentially chronic, but the mortality probably exceeds 30%.

Simulates bulbar paralysis.

MYOSITIS.—This condition may be suppurative or non-suppurative and represents an actual inflammation of the voluntary muscles. The symptoms are those of localized inflammation involving the muscles in a variable degree. They may feel doughy or in other instances hard, are tender and there may be slight edema of the skin overlying them or of the extremities involved. The diagnosis from trichinosis is only to be made by examination of the muscle fragments, though recent observations strengthen the assumption that trichinosis is usually associated with eosinophilia.

MYOSITIS OSSIFICANS PROGRESSIVE.—This begins with an

apparent cervical myositis which recedes and is followed by progressive induration and ossification, which may involve nearly the entire musculature. The disease is excessively rare and chronic.

Paramyclonus Multiplex.—See p. 32.

NEURITIS.—Inflammation of the nerves may be *single or multiple* and due to *toxæmia, extension of inflammation, actual germ invasion* (leprosy), *traumatism involving wounds, tears, concussion and torsion, pressure, and finally, to cold.* The toxæmias, whether due to mineral poisoning or acute and chronic infections are often associated with polyneuritis, the acute infectious diseases most frequently observed being diphtheria, erysipelas and leprosy, and of minerals, lead, arsenic and mercury. Beriberi is an acute infectious disease of which polyneuritis is the predominant and characteristic symptom. *Fatigue associated with chilling or prolonged exposure to cold and wet is a common cause of both local and polyneuritis; alcohol is also a potent cause.*

Infection, toxæmia, traumatism.

Fatigue and chilling

Alcohol.

Symptoms.—*Whether the neuritis be local or general, the symptoms are essentially the same, the differences being those of anatomic distribution. Pain is severe, persistent, boring or stabbing, and is associated with tenderness on pressure. It is usually most marked at the terminal points, but may be felt along the whole course of the nerve. Edema and congestion may be present over the same area, muscular power is impaired, motion is painful. Tactile sense is impaired or lost, formication and numbness are common, and actual paralysis follows, as does muscular atrophy, the skin becoming shiny, and the nails brittle and deformed.* Where the larger trunks are involved *the reflexes may be lost and the paralysis be typically flaccid.* It must be remembered that all degrees of inflammation are encountered as is illustrated by the behavior of the muscles in relation to the electric current. In some cases reaction of degeneration is complete and prompt, in others the response is normal.

Pain.

Tenderness.

Paresis.

Sensory disturbances.

Trophic changes and reflexes.

R. D.

ACUTE FEBRILE POLYNEURITIS.—This is merely a multiple neuritis characterized by an acute febrile onset exactly simulating that of the acute infectious diseases, the temperature being high as a rule. Subsequently a typical neuritis symptom complex develops taking the form usually of an *ascending paralysis* of varying degree, most often commencing in the feet, less often in the arms. *It may be limited to the extremities or affect the whole body even to the involvement of the diaphragm and other respiratory muscles.* Death is common in the severe forms, *but as a rule the patient recovers after an illness of a month or six*

Multiple neuritis.

Ascending paralysis.

Distribution.

Prognosis.

weeks. Residual partial paralyses are frequent, but seldom persist for more than a year.

Neuritis due to Alcohol, Lead or Arsenic.—*In this form the onset is usually gradual and preceded by numbness, tingling and neuralgic pain. "Foot drop" and "wrist drop" are common features, giving rise in the former case to the prancing or steppage gait. In alcoholic cases mental symptoms may be present,* but the history of the case usually points the way to a correct etiologic diagnosis, and it should be remembered that these paralyses chiefly affect the extremities, and that diphtheria shows a preference for the muscles of the eye and throat, lead for the extensors of the arms, arsenic for the legs and expecially the peroneal group. The distinctions are of course often disregarded.

Foot and wrist drop.

Prancing gait, in peroneal type.

Localization.

Beriberi.—This peculiar endemic epidemic neuritis causes a large mortality in Asiatic countries, is especially prevalent in the Malay Archipelago and a few important cases are encountered in this country usually from South American and West Indian ports or among fishermen on the Newfoundland banks. The cause is unknown, but the Japanese Surgeon-General has found that a marked diminution in the incidence of the disease follows the withdrawal of raw fish from the diet, and the substitution of unshelled for shelled rice. General hygiene undoubtedly plays an important part.*

Baron Takaki's great work.

Symptoms.—The symptoms are chiefly *those of neuritis* of greater or less severity. Edema is an important symptom varying from that of the slightest degree to a general anasarca. Palpitation and dyspnœa are also marked symptoms varying greatly in severity. A somewhat arbitrary classification is based upon the factors above named, there being a "mild form," in which all symptoms are slight, a "wet or dropsical" variety in which edema is the predominant symptom, the "dry form" characterized by predominant paralysis and relatively slight edema and the "pernicious form" in which cardiac symptoms predominate. *The diagnosis therefore depends upon the association of multiple neuritis with edema or marked cardiac disturbance in connection with the known prevalence of the disease, or the past residence, or associations of isolated cases.*

Mild, wet, dry and pernicious forms.

Diagnosis.

Recurrent Multiple Neuritis.—It would hardly seem that a separate place should at present be given to a polyneuritis showing a marked tendency to recurrence.

* Baron Takaki, in a recent conversation stated to the author that the mortality and incidence in the fighting forces had been reduced to an almost negligible factor.

Pressure Paralysis.—It is hardly necessary to do more than call attention to the various paralyses due to compression of individual nerve trunks. The common wrist drop of *musculo-spiral* paralysis and *circumflex* paralysis resulting from dislocation of the humerus are the most common. The former may result from crutch pressure, or pressure during deep sleep, especially "*in the open*," or may result from the position in which a patient is placed during a long operative procedure. (See also brachial plexus paralysis.)

Dislocation and crutch palsies.

General Points in the Diagnosis of Neuritis.—*Pain, tenderness on pressure over nerves, muscular weakness or paralysis, atrophy, early, though variable R. D., trophic disturbances and impaired tactile sensation following definite and known anatomic paths make the diagnosis of neuritis. Complete paralysis is necessarily of the flaccid type,* though *irritative symptoms in the form of localized twitching and spasm may be associated with a normal or partial electrical response; the steppage gait is characteristic of certain forms, inco-ordination, pupillary symptoms and lightening pains, absent; alcoholic* and lead *neuritis* are *usually* made evident by *the history* and *occupation, foot and wrist drop; and perhaps, delirium,* and in arsenical cases there is often a yellow skin.

Localizing features often striking.

Von Recklinghausen's Disease.—This interesting condition is characterized by a generalized neuro-fibromatosis, producing a remarkable variety of pressure symptoms referred to all parts of the body, patchy pigmentation of the skin and marked impairment of intellection.

A curious syndrome.

NEURALGIA.—See "pain" p. 68.

HERPES ZOSTER.—(Zona), (shingles). *This disease is characterized by violent neuralgic pain along the course of the superficial cervical, thoracic, sacral or abdominal nerves, or the branches from the Gasserian ganglion, associated with the appearance of crops of vesicles corresponding to the superficial nerve distribution and has its commonest seat over the lower thoracic of upper abdominal zone.* It is commonly unilateral, but may be bilateral and is supposed to be due to an inflammation of the ganglion cells of the posterior roots. *There is no disease which causes more exquisite pain and suffering, usually of a few days' duration, but sometimes extending over long periods,* and furthermore, troublesome ulceration may follow rupture and coalescence of the vesicles. The excessive severity and distinctly neuralgic or neurotic type of the pain and its distribution, and the typical rash makes the diagnosis clear, *but the pain may precede the herpes by many hours or even days and prove misleading, especially in the thoracic form.*

Usually thoracic and unilateral.

Extreme pain.

Delayed herpes.

PERIODIC TRANSIENT PARALYSIS.—This curious ailment

Recurrent, afebrile, transient paralyses.

is strikingly *hereditary*, often traversing five or six generations, and is characterized by the *rapid onset of paralysis involving the arms and legs, or both, sparing the face*, though sometimes implicating the regions supplied by the vagus and spinal accessory. *There is no fever, a slow pulse, general diminution, but rarely a loss of reflexes*, deep or superficial, and, *marked diminution of faradic excitability. These paralyses are transient*, lasting for only a few hours as a rule, but are strikingly *recurrent and periodic*, diurnal cases being noted. Like migraine the periodic paralysis usually ceases at the age of 50.

Prognosis.

RAYNAUD'S DISEASE.—(Symmetrical gangrene, local asphyxia). This mysterious hiemal vaso-motor affection of unknown causation assumes three forms, (1) *local asphyxia*, (2) *local syncope*, (3) *symmetrical gangrene*. Local asphyxia and local syncope in their mildest form may be seen in chilblains. In Raynaud's disease either emotion or trivial exposure to cold, or even gastric disorders are followed by *coldness, pallor of the surface of the fingers or of both fingers and toes.* After a variable period and in irregular order the affected members become *sharply congested and perhaps livid. Stiffness and discomfort or even severe pain may accompany the swelling. Symmetrical gangrene varies greatly in degree, is rarely extensive, and* usually a persistence of the extreme stagnation of the cyanotic stage results in *superficial necrosis* involving small areas or oftentimes *but one* finger or toe. *Sensation*, often diminished in the cyanotic stage, is lost over the gangrenous area which is black, icy cold and covered with blebs. *The area of apparent involvement greatly exceeds that of actual necrosis.* The ear is involved frequently, the tip of the nose occasionally, symmetrical areas on the arms, legs or trunk rarely. Cases of rapid, extensive and fatal gangrene have been reported and occur most often in children. Aside from the local symptoms there are no constant disturbances. Hæmoglobinuria, transient loss of consciousness, mental torpor and delirium are occasionally observed.*

Hiemal.

Three forms.

Exciting causes.

Lividity.

Gangrene.

Sensation.

Necrosis limited.

Occasional symptoms.

Diagnosis.—*The diagnosis, of course, depends upon the occurrence of symmetric localized syncope, cyanosis or gangrene without an assignable organic cause.*

*In one case observed by the author symmetrical gangrene of the ears of the hiemal type had resisted treatment for years, but recovered promptly when the cause was found. The patient was taking large quantities of morphine by the hypodermic method, the skin over large areas of the trunk and limbs being thickened and infiltrated through the use of a dirty syringe. As withdrawal was resisted the patient was told to take the drug by the mouth, since that time (14 years) there has been no recurrence of the gangrene.

ERYTHROMELALGIA.—This rare condition unlike Raynaud's disease is essentially a summer ailment and is characterized by *superficial congestion, swelling and burning pain* in the feet or hands usually the former, and worse at night, the pain being severe and increased by the dependent position. It is chronic and irregularly recurrent, often associated with headache and vertigo, and occasionally with Raynaud's disease.

One of the four bastard syndromes.

ACROPARÆSTHESIA.—This symptom complex is closely allied to angio-neurotic edema and is characterized by itching, burning, prickling and pain in the fingers and toes. Heat, cold and injury are apparently causative factors; constipation and gastric disorders almost constant, and occasionally there is rigidity of the fingers. It is hardly worth separate classification.

Subjective.

INTERMITTENT JOINT EFFUSIONS.—Closely allied to angio-neurotic edema is this rapidly produced but painless swelling of the joints. It lasts for only a few days, but is liable to recur.

ANGIO-NEUROTIC EDEMA.—This vaso-motor condition seems closely related to urticaria and, like it, is usually associated with digestive disturbances. Exposure to cold and emotion may be associated with it, but it often appears without apparent cause. It *is essentially a sudden and transient swelling, affecting usually the hands, feet, genitalia, or any portion of the face,* rarely the lips, tongue or even the glottis where it may produce dangerous or fatal obstruction. As in Raynaud's disease associated hæmoglobinuria has been noted. *It may be periodic but is more often irregularly recurrent.* The *gastro-intestinal symptoms may be severe and associated with vomiting and colic,* or, extremely slight. Osler reports a case in which the whole arm was swollen. Aside from the rare involvement of the glottis the disease is as unimportant as it is obscure.

Usually trivial.

Occasionally serious

FACIAL HÆMIATROPHY.—This incurable and obscure condition, most frequently developed in childhood, rarely in the adult, is a progressive atrophy involving all structures on one side of the face. It is peculiar in that it affects first the skin and superficial tissues, then the bone and lastly the muscles. Loss of the hair and teeth and hæmiatrophy of the tongue and palate usually occur, but bilateral atrophy is rare and sensation and the electric response of the muscles is usually unaffected. It is probably not due to disease of the trigeminus and as is generally held should not be confounded with congenital asymmetry of the face. In direct contrast is unilateral facial hypertrophy in which every symptom is reversed.

Peculiar line of march.

Alternatives in heredity.

Exciting causes.

Age.

Sex.

Senile cases.

EPILEPSY.—This *psychoneurosis* depends to an extraordinary degree upon heredity, but in predisposed individuals may apparently arise from emotional shock, acute toxæmias and sometimes from remote and purely reflex causes. One variety arises from direct irritation due to injury, recent or remote, or forceps injuries in childbirth, *and one-half the cases occur in the first five years of life. It is so rarely a primary lesion in the adult as always to excite a suspicion of organic* disease. *Sex.* The incidence is nearly equal in the two sexes. Of *inheritance* it should further be said that *it is not so much direct as equivalent* (see page 47). Parental intemperance, for example, is an important factor, syphilis and alcoholism common and direct causes. *Attacks very similar to true epilepsy occur in arterio-sclerosis*, which condition probably accounts for the cases met with in old people.

Divisions.—We speak of "*major epilepsy*" (grand mal), "*minor epilepsy*" (petit mal), "*Jacksonian epilepsy*," and, "*psychical epileptic equivalents.*"

Auræ.

Grand Mal.—*Auræ and various subjective sensations precede an attack.* There may be auditory, visual, gustatory and olfactory paræsthesias, discomfort in the epigastrium, intestines or rectum, or peripheral sensations usually referred to the hand. *In other cases the aura is motor*, the patient turning rapidly, running for a short distance or twirling about on the toes. *The actual onset* is sudden, sometimes preceded by the so-called epileptic cry. A fall is likely to produce injury, a matter of importance in differentiating hysteria, and, *the condition is one primarily of tonic spasm*, the patient being livid and usually in a distorted attitude; *after a few seconds* follows the stage of *clonic convulsions*, at first slight, but increasing rapidly in violence and affecting the ocular and facial muscles as well as those of the body and extremities. The patient froths at the mouth, is likely to bite the tongue, or pass urine or fecal matter involuntarily. *The duration of this stage is variable*, but it *seldom lasts over two or three minutes* and is followed by coma, succeeded by a deep sleep lasting for several hours if undisturbed, the patient wakening with slight mental confusion or perhaps headache. *The attacks vary greatly in frequency, usually tending to increase with the lapse of time. They may occur only at night* and the patient may be and often is, entirely unconscious of their true nature, a point to be remembered in case-taking.* In the so-called "*status*

Onset.

Seizure.

Tongue biting.

Incontinence.

Coma.

Increasing frequency.

Nocturnal.

* In one such case observed recently there was nothing to mark the attack in the morning but a slight persistent injection of the facial capillaries, the patient feeling perfectly well, with unclouded mind.

epilepticus" there is fever, rapid respiration and increased pulse rate, accompanying a succession of attacks without *persistent* unconsciousness. The reflexes are sometimes absent, more often increased and accompanied by ankle clonus. *The urine if not passed during the attack* as is usual, is *passed in quantity after it. Post-epileptic paralysis and aphasia* are occasionally seen but are ordinarily transient. *In repeated seizures mental deterioration may be marked and post-convulsive mania may assume a dangerous form and become of medico-legal importance in murder cases.* So also these unfortunates may be arrested for indecent exposure due merely to mental disturbances, characterized by various automatic actions of which the patient has no consciousness or recollection.

Polyuria.

Mental changes.

Spurious exhibitionism.

Petit Mal.—*This interesting condition lacks the convulsive features of epilepsy, though in a majority of instances grand mal develops, either wholly replacing or alternating with the minor attacks.* Amongst the various forms may be mentioned *simple incoherency, acts of automatism,* usually of short duration, *sudden falls, with or without transient loss of unconsciousness, sudden jerkings, tremor or subjective sensations of various sorts, mere interruption of conversation, and a peculiar absent mindedness.* During such attacks the patient is likely to turn suddenly pale, there may be fixation of the eyes and relaxation of the grasp but auræ seldom occur.

Convulsions absent.

Curious manifestations.

Jacksonian Epilepsy.—This is the reverse of petit mal, *consciousness being retained early in, or throughout, the attack and convulsive seizures present.* One must admit possible sensory equivalents but direct cerebral irritation is nearly always present. *The spasms are usually orderly* and *follow motor areas of the cerebrum, but may be limited to one tract such as the face and leg.* Partial epilepsy, moreover, often becomes general with the lapse of years, old injuries, tumor, meningitis, cerebritis, uræmia, abscess, hemorrhage and sclerosis are the usual causes and post-hæmiplegic epilepsy is of this type.

Petit mal reversed.

Motor tract spasms.

Epileptic Equivalents, Psychoses.—Hallucinations of hearing, narcolepsy (profound sleep), and sometimes, certain cases of somnambulism, seem to replace the convulsive stage of epileptic seizures, and in some instances, certain moral deficiencies are also prominent, often amounting to an entire change of character or the development of traits or habits abhorrent to the patient in a condition of health.

Peculiar and important.

Differential Diagnosis.—If an attack of major epilepsy be seen little doubt can be entertained as to its nature, though *only by an examination of the urine can uræmic convulsions be positively differentiated.*

Uræmia and hysteria.

Hysteria offers many difficulties, but is seldom accompanied by tongue biting, involuntary passage of urine, and *never by the absolute unconsciousness presented by the epileptic.* Hysterical patients choose a safe place to fall and the movements are usually disorderly and to a certain extent purposeful and struggling rather than jerking; moreover, the duration of the hysterical attacks is longer and often promptly relieved by sharply applied pressure over the ovaries.

Jacksonian epilepsy is unmistakable, though its cause may be obscure; petit mal, often beyond the range of absolute diagnosis, its recognition *depending upon the coincidence of transient unconsciousness, vertigo, and perhaps automatism.*

Etiology.

Precocious children.

Unwise parents.

Secondary factors.

Reflex course.

Age and sex.

CHOREA.—(St. Vitus's dance, Sydenham's chorea). This is a disease of unknown causation apparently depending largely upon an inherited or acquired neuropathic temperament, Precocious, imaginative, excitable children are peculiarly liable to the disease, especially if, as is so often the case, they are pushed ahead in their studies or encouraged to develop any unusual mental traits at an unduly early age. *Overforcing is the chief cause of chorea, and the elements of shock, worry, grief, etc., are merely secondary factors in causation.* Seizures due to reflex *irritation* are rarely seen but may occur in connection with various conditions affecting the gastro-intestinal tract, sexual organs and more often as a result of *naso-pharyngeal adenoid* growths. The disease *predominates in females* between the ages of 5 and 15, *is less often seen in the well-to-do, and seems to bear some relation to rheumatism and a more direct one to heart disease.** It is often difficult to decide whether

Rheumatism.

Heart disease.

rheumatism preceded or followed the development of the disease, and the same difficulty is encountered in heart disease which is by many believed to be the cause of chorea (the so-called embolic theory). *It is certain that about* $\frac{2}{3}$ of the cases *show valvular murmurs,* but it is by no means proven or even rendered probable that a resultant embolism is the cause of the disease. If we assume, however, a neuropathic constitution, to which is added rheumatism or a valvular heart lesion with its attendant disturbances of circulation, it is easy to see that in a sense the latter might be causative. The author believes that beyond such an assumption there is no adequate basis for any of the present theories concerning the etiology of chorea.

Symptoms.—*Involuntary, inco-ordinate, characteristically jerky movements, unilateral or bilateral, localized or general, and associated to a*

* In Osler's analysis of 554 cases only 15 $\frac{8}{10}$% showed a clear history of acute or subacute arthritis.

varying degree with excitability and emotional disturbances, characterize chorea. Though difficult to describe it is never forgotten when once seen, nor does it offer any difficulties to the diagnostician save in its rarest forms. According to its severity we recognize a *mild and severe form*, to which may be added a *third, the maniacal chorea*, which is associated with delirium. The *predisposing factors* have already been considered and the exciting causes of an attack are often of an emotional sort.

A clean-cut clinical picture.

Varieties.

Symptoms.—*Premonitory symptoms* take the form of *increased nervous irritability*, perhaps *unusual wilfulness*, and a *general restlessness.* As the irregular movements begin, the apparent clumsiness of a child, associated perhaps with unusual disobedience, may bring about undeserved punishment which may precipitate the attack. *The face and arm are most commonly involved*, but in severe cases the whole body may participate. *Disturbance of speech occurs in about one-fourth of the cases*, varying greatly in degree *and the facial grimaces and jerking, inco-ordinate and bizarre movements of the extremities involved are absolutely distinctive.* If the legs be affected a peculiar *jerky hitching gait* appears, and there may be motor weakness in the leg or arm. Occasionally there is crossed chorea, still more rarely one encounters the most severe type, maniacal chorea. *Mental disturbances of marked degree are unusual* yet intervals of delirium or temporary hallucinations sometimes occur. Practically all of the other associated symptoms would seem to be dependent upon rheumatism and endocarditis as factors. *The heart symptoms* are chiefly those of functional or organic murmurs, the latter being present in about 50% of all cases and two-thirds showing a murmur of some sort. The ordinary *hemic murmur* with its maximum at the second pulmonary interspace is of little moment, *and other functional murmurs may be associated with the accelerated heart action almost invariably present. The common murmur is systolic and apical* and the differentiation between the functional and organic lesion must depend upon the application of the usual diagnostic rules. *Evidence of secondary dilatation and the presence of a loud harsh murmur indicate an organic mitral lesion.* Arsenical pigmentation may occur, but fever, purpura, urticaria and joint symptoms have probably no direct relation to the disease itself, but rather to the digestive disturbance and rheumatism with which it is so frequently complicated.

Misunderstood clumsiness and "badness."

Speech, gait.

Rare mental symptoms.

Heart signs.

Murmurs.

Cutaneous signs.

Course.—Although showing a distinct tendency to recurrence (50%), the individual attacks seldom last over two or three months, though

rarely they may extend over years, particularly as slighter manifestations. The death rate during the attacks is almost negligible.

Differential Diagnosis.—It should be remembered that *choreiform movements* are increased by excitement or obstruction, *entirely cease during sleep*, whether natural or produced by drugs, *and, that neither sensibility nor, usually, reflex activity is affected. Chorea electrica* is merely a variant of chorea proper characterized by lightening-like movements. *Huntingdon's chorea is hereditary, appears usually during the third decade and is characteristically constant and progressive. Mental deterioration is marked and it usually progresses to terminal dementia. The movements themselves are slower, the gait more swaying and the disease should have a single descriptive name.**

Hereditary chorea.

Friedreich's ataxia.

Friedreich's Ataxia. Nystagmus, scanning speech, scoliosis, talipes, the slow movements and clear evidence of heredity make the diagnosis clear.

Hysteria.

Hysteria. Rarely, cases may exactly simulate chorea, but usually the movements are less jerky and irregular, more rhythmic and there are associated stigmata which make the diagnosis easy. Rhythmic chorea of hysterical origin produces orderly movements of the muscle groups involved, for example, the *salaam convulsion.*

CONVULSIVE TIC.—(Habit spasm). This usually takes the form of localized facial spasm associated with head movements or the raising of a shoulder, though often the spasm affects only the eye or the mouth, or more commonly both. It is a somewhat common, transitory and unimportant condition.

IMPULSIVE TIC.—(Gilles de la Tourette disease). This is characterized by *involuntary facial, brachial or general muscular movements, often violent, in association with marked disturbances of speech and sometimes of mentality. Articulation is explosive even to incoherence, mimicry may be present, usually in the form of repeated utterance of words heard or of actions seen, or, the frequent repetition of obscene words may accompany the spasm.* The *mental disturbances are similar, taking the form of obsessions or fixed ideas.* Every action may be associated with an impulse to count to a certain number or to touch a certain object.

Speech and gesture.

Mimicry.

PANDEMIC CHOREA.—This group of diseases, more closely allied to hysteria than chorea, is chiefly of historic interest, although rare instances are seen at the present day. The tarantella of the composer was suggested by the remarkable religious manifestations epidemic during the middle ages. These were characterized by the wildest ex-

The tarantella.

* It is variously described as "megrim," "megrum," "senile," "chronic progressive," "adult hereditary" and "chronic," chorea.

citement, violent gesticulation, and dancing and leaping to the point of exhaustion.

SALTATORY SPASM.—This is a transient or chronic condition characterized by *sudden violent contraction of the leg muscles upon any attempt to stand.* Marked mimicry of words and actions may be present, and heredity plays a part.

PARALYSIS AGITANS.—(Shaking palsy, Parkinson's disease). Occurring more frequently in men than in women and seldom under the age of 40, this disease possesses no definite etiologic factors, nor has it any characteristic pathology unless it be a premature senility of a cerebro-spinal type.

Symptoms.—The chief symptoms are *tremor, rigidity, weakness and a peculiar attitude and gait.* The disease, though insidious, is easily recognized when fully developed. *The tremor* chiefly affects the hands and feet, thumbs and forefingers showing the so-called "pill rolling" motion; the toes are less involved, the chief tremor in the lower extremities being in the ankle joint, and *head nodding*, if present is usually vertical, more rarely rotatory. Emotion increases tremor; voluntary movement may or may not check it, and it is absent during sleep. *Rigidity* is shown in slow and awkward movements, and weakness may be the earliest symptom but often appears only after exertion. *The face is immovable and expressionless, the eyebrows raised and the attitude and gait* become characteristic as the disease advances. The *patient stoops with bent head, with the arms* held away from the body and *flexed at the elbow* and the fingers bent. The *voice is high* pitched and *the speech* curiously hurried though perhaps primarily hesitating. In walking *festination* is observed, the stooping patient apparently trying to overtake his centre of gravity (Trousseau) and if thrust backward the same short hurried steps are taken and the patient will fall if not supported.

Bread crumbling tremor.

Rigidity.

The mask-like face.

Attitude, voice and speech.

Festinant gait.

Propulsion and retro-pulsion.

Diagnosis.—The developed disease is unmistakable and a diagnosis may be made on the basis of rigidity, weakness and attitude alone. It could only be confounded with post hemiplegic tremor from which the history will usually at once distinguish it.

HYSTERIA.—*The fact that one is dealing with a genuine disease should never be forgotten in relation to hysteria, and further, that it is a disease equalled only by syphilis in the protean nature of its manifestations.*

A disease.

Etiology.—*Heredity and early training are the most important factors in hysteria.* Indulgence of whims, the development of the emotional

Heredity and early training.

side by excessive sympathy, the failure to demand of the child or young adult self control and consideration for others are factors that can seldom fail to create an hysterical temperament, particularly if the body be relatively feeble, the intellect precocious and the *neuropathic hereditary taint marked.* In such persons nervous instability, lessened will power and suggestibility, are often evident early. Grief, joy or even the minor worries of life produce hysteric seizures and the condition is aggravated and increased by the injudicious petting, solicitude and indulgence which such unfortunates receive at the hands of parent, husband, or even the physician. On the other hand the condition, when developed, must not be regarded as one depending upon the unassisted volition of the individual, or wholly subject to her control, but rather as an actual disease to be ameliorated and perhaps removed by a treatment which, though kind, is *firm, persistent, insistent and lacking in every element that enters into the upbuilding of the hysterical temperament.*

Harmful sympathy.

Treatment.

Young women chiefly.

Age, Sex and Race.—The disease occurs for the most part between the ages of 15 and 20, and almost exclusively in women, cases of hysteria in the male being the rare exception in this country. It rarely occurs in children under 10 and adults over 60. Southern races yield many examples as compared with the more Northern. *Relation to Disease. It is found* in *combination with and as a result of actual disease far more frequently than is usually supposed, malnutrition and anæmia,* lead and arsenic poisoning, unrecognized dyspepsias, gastric or intestinal, reflex irritation, naso-pharyngeal obstruction, or diseases of the pelvic organs, movable kidney, chronic appendicitis and unrecognized gastric ulcer being some of the causes encountered by the author. Physicians generally err about as often one way as the other, failing on the one hand to recognize an underlying organic cause in certain cases, and on the other construing pure hysteria as the organic disease which it simulates. *Sexual excess or sudden deprivation in certain instances is most important,* though aside from those showing erotic posturing, etc., during attacks, the hysterical woman is usually sexually indifferent and the genesic sense *may* be abolished through anæsthesia.

Often associated with organic ailments.

Sexual factors.

Symptoms.—These may be present in almost any portion of the body and consequently almost any disease may be simulated. *Motor symptoms* may be paralytic, paretic or spasmodic. *Paraplegia and monoplegia* are more common than *hemiplegia. Paralysis* is usually incomplete, muscles may show disuse atrophy, *but the electrical reactions are normal,* the knee jerks seldom diminished, usually increased

Hysteric stigmata.

and equal, and a paralyzed leg is usually dragged limply along. *Urinary retention may be seen*, true *incontinence never*, true ankle clonus is absent, pseudo-clonus sometimes present; *aphonia* is frequent but the paralysis is seldom complete. *Ptosis* is usually double, often with an overactive orbicularis, but the muscles of the eyeball are seldom affected, though conjugate deviation *rarely* occurs. *Both tonic and clonic spasm* may occur, particularly after injuries and both paralysis and spasm may be associated with *anæsthesia*. *Pseudo-contractures, persisting during sleep but disappearing under general anæsthesia*, and fine *tremor* or regular and irregular muscular contractions and even edema are encountered. *Hystero-epileptic* seizures are common among the Southern races, but their differentiation is seldom difficult, though their various phases (i.e., prodromal, epileptoid, clownism, passional, delirious), may vary greatly in intensity or appear in an isolated form. *Hyperæsthesia* is especially marked in the hysteric zones, i.e. *the hypogastrium and lower spinal* and *inframammary regions* but it may be *visual, auditory or olfactory*. *Anæsthesia and more often, analgesia* may be associated with loss of response to heat and cold and impaired muscle sense. *Both motor and sensory paralyses affect the left more often than the right side, but sensory symptoms may involve the entire body, including the mucous membranes* and in hemianæsthesia they are characteristically limited by a well defined border representing the median line of the body. The stocking, garter, glove, sleeve or drawer leg forms are seen, as are irregular or disseminated islets of anæsthesia conforming to neither nerve nor segment distribution while they may capriciously shift their seat or boundaries after a seizure or without apparent cause. They may also take the form of *concentrically* contracted visual fields usually unequal and sometimes unilateral or *hysterical color vision* (see page 555). *The loss or perversion of special senses* and *loss of sensation is actual and not imaginary in all true hysteria*, and the patient is usually unconscious of the latter. Absolute blindness or deafness is rare and monocular diplopia is a rare symptom. Mental symptoms range from mere hysterical laughing and crying, to amnesia, loss of will power, impressionability, somnambulism, and even the simulation of actual insanity. The pharyngeal reflex is lost in 90% of the cases of hysteria as is frequently the plantar reflex. *Autohypnotism* and *catalepsy* are among the rare conditions, the reflexes being lost in the latter condition.

Contractions rather than contractures.

Some form of anæsthesia seldom absent.

Diagnosis.—Most cases are evident to any trained clinician and in those simulating organic disease of the spine or cord some difficulty

may arise. The *normal or increased knee jerk, absence of urinary incontinence and true ankle clonus*, the *normal electrical reactions*, absence of true atrophy, and the *active resistance* of spasmodic contractures, i.e. the evident active resistance offered the examiner's hand, are quite unlike true spastic contraction. *The onset of symptoms is moreover generally coincident with some violent emotion, shock, or injury.* The disappearance of contractures during anæsthesia or sound sleep is also important and the minor seizures may instantly disappear under sharp ovarian (hypogastric) or supraorbital pressure. Disseminated sclerosis presents oftentimes a strong resemblance, but tremor is absent or markedly lessened when the patient is at rest, continuous in hysteria.

Prognosis.—The prognosis in any case depends upon two factors, the ability to undergo a proper course of treatment and the physician's success in obtaining the co-operation of sensible and intelligent family members. *In many instances surgical or medical treatment directed to the cure of underlying organic disease or the removal of reflex irritation completely removes the symptoms.*

NEURASTHENIA.—This ill-defined and protean ailment depends for its development upon much the same conditions as have been described under hysteria, hereditary predisposition being marked in most instances but the factors of *over-work, mental strain and past disease* are more prominent. *Sexual excess, perversion or sudden deprivation* cuts a considerable figure, but *drug habit is a sequence* rather than a cause.

Common causes.

Symptoms.—*The fundamental symptoms are those of an unstable and irritable nervous system. Hypochondria* is a frequent feature and more or less marked mental depression, either constant or occurring at intervals, is the commonest symptom. As a result, minor worries or physical ailments are magnified or even created out of nothing, and a loss of interest in those matters which have formerly been an engrossing life factor, inability to concentrate the attention, lapses of memory, periods of irritability, faultfinding or passion may replace a kindly jovial temperament. *Egotism and introspection united make the patient a bore to his friends, family and physician* and with this goes usually an utter lack of appreciation of the equal rights of those about him. If, as usually happens, *insomnia, cerebral pressure* and *headache* appear, the fear of insanity may be uppermost in the patient's mind. Such are sometimes dominated by *fits of restlessness* and give way to *emotional crises.* Threats of suicide are common, and attempted suicide not infrequent. Many shun society, others fear

Mental depression.

Irritability.

Egotism.

Headache and insomnia.

Emotional crises.

to cross open spaces alone, yet others are terrified in passing high buildings or engines in operation, and may even show the same causeless anxiety in every relation of life (pantophobia). *The local manifestations relating to the heart, lungs, stomach, kidneys, intestines*, etc., are so well known as to need no special description. *Practically every organ may be the focus and apparent source of symptoms closely related to those of organic disease*, and it should be said that *here as in the case of hysteria the utmost care must be observed in distinguishing between neurasthenic states sequential to disease and removal only by attention to the primary cause*, and *those which represent a neurasthenic state only*. From personal observation the author has become convinced that *a large number of so-called neurasthenics are actually suffering from organic disease to which a neurasthenia is secondary, and it may further be affirmed that the great success attending rest cures is in some measure due to the wide therapeutic range of rest and its associated measures for improving nutrition in the domain of general medicine.*

The Special Senses.—Increased sensibility to pain and general hyperæsthesia find their local expression in the special senses. Neurasthenic asthenopia is extremely common and acoustic disturbances hardly less so. *The Muscles.* Muscular weakness even to complete loss of power is said to be possible but in most instances is a purely subjective nervous phenomenon. A patient who feels utterly exhausted may engage in golf, curling, horseback riding or some similar pursuit and find his fatigue disappear with exercise or preoccupation. *Vaso-motor Phenomena. Relaxation of the peripheral arteries together with the irritable heart* so frequently observed, may give rise to subjective sensations of throbbing or auditory hallucinations of the same character, and, indeed, may produce *visible peripheral pulsation and a pulse not distinguishable from that of aortic regurgitation.** Capillary pulse is frequently observed and Osler reports cases of venous pulsation. The well known throbbing of the abdominal aorta leads often to an erroneous and usually *inexcusable* diagnosis of aneurism, and local flushing and sweating and coldness of the extremities are common symptoms. *Sexual neurasthenia forms the basis of the most troublesome of the complaints that come to the physician.* Nervous impotence may occur, troublesome sexual irritability is not uncommon and ovarian and testicular tenderness is often wholly neurasthenic. *Symptoms simulating diseases of the cord*, such as localized spinal tenderness, intercostal neuralgia, subjective fatigue, paræs-

Subjective weakness.

Misleading pulsations.

The physician's bête noir.

* Such cases are more frequently associated with a combination of neurasthenia and decided anæmia.

thesia and visceral neuralgias may be marked and inco-ordination is occasionally well developed. This, however, is a relatively rare symptom

Vertigo.

and more often *vertigo*, usually transient, is experienced. *A very important sign in this connection is a dejective articulation precisely similar to that of the early stage of paretic dementia.*

Speech.

This is not in the author's experience an uncommon symptom though rarely existing in a marked degree. *Reflexes*, both superficial and deep, are usually increased but *pupillary reflexes are normal*, though occasionally the pupils themselves are dilated or unequal.

Reflexes.

Diagnosis.—A consideration of the etiologic factors in connection with the foregoing symptoms is usually quite sufficient for a diagnosis. It will be readily seen that *insanity and paretic dementia in their early stages present almost typical neurasthenic symptoms, as indeed do certain of the degenerative diseases of the brain* and *cord.*

Danger of misinterpretation.

The physical examination is of great value in the exclusion of organic disease but not otherwise. The absence of true hysteric paroxysms separates it from hysteria, nor do such patients present the more marked hysteric stigmata. A drug habit is often over-looked and the same is true of petit mal. As a matter of fact, however, 99 cases out of any 100 presenting the symptoms detailed under this head are neurasthenics and the progress of the ailment in the case of insanity and paresis, together with the more pronounced earlier symptomatology, the disturbance of reflexes and other well known signs of organic diseases of the cord, such as locomotor ataxia and multiple sclerosis, leave little room for error. The diagnosis of neurasthenia rests, therefore, upon the basis of a thorough and careful examination of every case and such will almost invariably exclude any of the confusing disease factors and often relegate a supposed primary neurasthenia to an expression of anæmia, cardiac, renal, gastric or other organic disease.

See a primary cause

TRAUMATIC NEURASTHENIA AND HYSTERIA.—(The Traumatic Neuroses of Oppenheim).—(Railroad spine, railroad brain). No more difficult task presents itself to the physician than that involved in an opinion regarding the effect of injuries as represented by purely subjective or indeterminate objective symptoms and too often involving a large element of unconscious or deliberate deception. To do justice alike to an injured individual and the harassed and much bled corporation is at times well nigh impossible.

A difficult problem.

Important Features of Traumatic Cases.—Aside from out and out malingering one may encounter one of four reasonably distinct *clinical* types, viz.:—(1). *Those in which a pure neurasthenia is mani-*

Clinical types.

fest. (2). *Cases markedly hysterical.* (3). *Cases exactly resembling the first two groups but developing after a variable period actual disease of the brain or cord.* (4). *Cases of manifest injury.*

Neurasthenic Type.—This differs in no particular from ordinary neurasthenia save that the morbid introspection and mental depression relate to the accident and its supposed effects.

Hysteric Type.—This is merely a neurasthenia with hysteric stigmata added, the emotional side being pronounced.

Symptoms Commonly Presented.—Headache, insomnia, vertigo, subjective weakness, mental and nervous irritability and instability are marked, mental depression is common and may reach actual melancholia. Pain in the back is almost invariable, the reflexes are increased and the impaired digestion and lack of appetite, together with the mental depression and anxious, drawn countenance, often lend a misleading facial element of suffering, emaciation and pallor. The tongue is usually coated and constipation is common. Numbness and tingling may be present in either the hysteric or neurasthenic types, but in the former may co-exist with either a mere limitation of the visual fields or an achromatopsia and a marked hysteric or emotional tremor is not uncommon. The pupils are often dilated and may be slightly unequal. *Circulatory disturbances* are common and tachycardia, marked cardiac irritability, arrhythmia and vaso-motor disturbances often persist for long periods. Subjective sensations of heat and cold, flushing, excessive perspiration or recurrent sweats, variations in the urinary secretion and the like frequently occur. Menorrhagia, amenorrhœa, sexual neurasthenia and loss of memory may be added to the long list of symptoms usually representing purely functional disturbances.

Neurasthenic and hysteric.

Physiognomy.

Paræsthesias.

Pupils.

Tremor.

Heart.

Vaso-motor and miscellaneous.

From personal observation the author believes that no line can be drawn between "traumatic neurosis" and traumatic neurasthenia or hysteria, for while at times cases arise that fit the syndrome first named, in many others there is a blend of signs that hopelessly blurs any sharply drawn boundaries and the element of deliberate or unconscious exaggeration too often fostered by alarmist opinions and vague theories on the part of the over-sympathetic physician pervades and weakens the whole diagnostic structure. He also doubts the *frequency* of "traumatic neuroses" so called after trivial falls and injuries when not connected with claims for damages, and has more than once found them properly attributable to pre-existing disease of the heart or blood vessels, usually a myocarditis. In those predisposed to or actually suffering from neurasthenia or hysteria those conditions may readily

Effect of trivial injury.

How explained.

be excited or intensified by slight trauma or excitement. There are probably but three *actual* conditions, viz.:—(1). *Genuine injury to and actual lesions of the brain or cord.* (2). *Pure neurasthenia or hysteria, and* (3) *pure or adulterated malingering, usually for revenue.* The *diagnosis* must depend upon the discovery of symptoms purely subjective or of the neurasthenic or hysteric type on the one hand, or, *definitely* objective upon the other. Lacking objective signs after several months one is *reasonably certain* that the case will recover after settlement. With the signs of organic disease and only in their presence is the physician justified in a lugubrious prognosis on or off the witness stand. In fully 80% of such cases, examined by the author, in which suit was pending, the element of exaggeration was *evident.* In hardly any of those injured in a similar manner but without liability on the part of a corporation have there been any symptoms save those of direct injury such as bruises and sprains, neither has the pain in these been persistent over long periods, nor the mental and nervous symptoms, *save in exceptional instances, even in the cases associated with great primary mental shock.* Indeed it must plainly appear to anyone experienced in these matters that *the after symptoms in railroad injuries are remarkably in consonance with the degree of mental shock experienced at the time.* Finally there is the group of cases which are truly surgical or relate to actual visceral lesions and of fracture of the vertebræ, sprains, hemorrhage into or about the cord, myelitis and meningitis and are directly diagnosticable, though in rare and deplorable instances the *delayed* development of organic disease may result in an injustice to some honest suitor.

Actual conditions.

Prognosis.

Evident exaggeration.

Sprains, fractures, etc.

MALINGERING.

Until medicine becomes an exact science the physician will do well to give the benefit of any doubt to the supposed malingerer and avoid measures savoring of harshness or cruelty, or the giving of injurious testimony in a court of law, save when the case is clear and the imposture of a particularly barefaced or injurious nature.

Mistakes common.

The chief sufferers from feigned diseases are railway, street car and accident insurance companies, but the evil extends to the army and navy, pension bureaus, and even to the home, and every physician must meet with numerous cases whatever may be his line of practice.

Chief sufferers.

No one class of diseases has any monopoly of this form of deception, though the nervous system lends itself most readily to the needs of the impostor, while on the other hand presenting more traps and pitfalls

and in certain directions more difficulty in simulation than general diseases.

The basis of the most serious types of malingering or imposture is the dollar and the lengths to which people will go in their attempts to defraud accident insurance companies especially is almost incredible. For many years these companies paid a large indemnity for the loss of the left hand and the number of claims incurred was so enormous as to lead to a reduction of the indemnity by one-half, following which the claims of this class fell off over 80%.

Malingering for revenue.

Striking illustration.

Men will deliberately shoot themselves or place a hand or foot under the wheels of a moving train for the sake of the small amount of money represented by accident indemnity and similar examples of self mutilation are found in the countries where military service is compulsory or in the case of soldiers tired of service and desiring a discharge; the common form being the mutilation of the trigger finger as furnishing the readiest and slightest injury incapacitating for active service. The minor frauds in this connection are consequently encountered by every physician and the unscrupulous man with an unrecognized but readily sprained flat foot or the still more fortunate possessor of a shoulder or hip that he can dislocate at will is reasonably sure of realizing something from his infirmity. Furthermore, as is now well known, certain unscrupulous attorneys and equally unscrupulous physicians of the large cities form a criminal class known as "ambulance chasers." Such scoundrels will not only follow up cases of injury and suggest lawsuits but will furnish witnesses ready made, sworn physicians' statements of any desired character, and any other bit of criminal machinery needed in the given case.

Army service.

Flat foot and hip joint cases.

Ambulance chasers.

A Curious Case of Simulated Injury.—Amongst the many instances of professional malingering one will suffice for illustration. A certain individual, following the usual custom of the criminal, took one special line as his own and the street car company as his object of attack. His procedure was simplicity itself; he would enter a street car carrying a cane bearing on its tip a screwdriver and while sitting in the car would quietly elevate a floor screw of the seat in front of him, then he would rise hastily to leave the car, ostensibly trip over the screw head and in falling receive an apparently severe and painful injury. He would then summon a carriage and be driven rapidly home where he would take a tack hammer and after covering the knee, elbow or ankle joint with a few layers of cloth would pound the point selected until he had produced the necessary appearance of a severe joint injury

Malingering extraordinary.

after which he summoned a physician. It is said that this went on for years and that the man received in damages from different corporations a very decent fortune. As regards deliberate mutilations of the more serious type it should be remembered that the victims are usually urgent in their demand for amputation, frequently suggesting it and often insisting upon it. Such are usually holders of recently issued accident policies, and the mutilation is ordinarily just sufficient to come within the terms of the contract and seldom involves the most useful member; the *left* hand or foot being usually selected.

Suggestive facts.

Classification.—Disease or injury may be (a) wholly *spurious and non-existent, or,* (b) *actual, but either self produced, factitious or deliberately exaggerated or aggravated,* (c) *the deception may be an innocent one due to a genuine belief in its serious nature* on the part of the patient which opinion is usually fostered by friends and family and too often by an over-sympathetic physician.

Non-existent.
Factitious.
Exaggerated.
Imaginary.

SOME OF THE COMMONER FEIGNED STATES.—Anæmia.—By taking a nauseating substance pallor is readily achieved but the absence of anæmia is readily demonstrated by testing the blood.

Angina Pectoris can be successfully simulated by one familiar with the subjective symptoms by swallowing tobacco and taking drugs which produce a disturbed heart rhythm, pallor and clammy skin, and simulating the paroxysmal seizures. It requires, however, a good actor. The tobacco or other drugs may be taken by the rectum and palpitation may be caused by extreme compression of the abdomen, indeed Herold states that hypertrophy may thus be induced, which statement we would be inclined to doubt.

Aphonia.—Here the one reliable test is the volubility of the patient during the stage of excitement or the after stage of recovery from an anæsthetic. No condition is more readily simulated.

Asthma of the spasmodic type cannot be successfully simulated nor can the severer form of dyspnœa because of the absence of cyanosis save in the case of a very patent holding of the breath.

Atrophy of the Extremities.—A certain amount of atrophy may be induced by disuse and increased by the use of tight bandages.

Blindness.—Amaurotic blindness is easily feigned by instilling atropia and is difficult to detect unless the patient can be thrown off his guard and carefully observed for a considerable period. Simulation of blindness, however, usually involves one eye and is oftentimes a mere exaggeration of an existing though trivial defect in refraction. A

pupil reacting normally to light excludes most forms of blindness but in all, the testing of the visual field will often reveal deliberate deception and a simple test consists in holding a pencil close to the sound eye and asking the patient when its tip is lost to his vision; he may still see it after it is passed behind the nose or deny it while it is manifestly in the range of the normal eye. If asked to look at an object held in his own fingers a malingerer feigning total blindness will often pretend that he cannot get the direction of the object while those actually blind will look at it, judging its position without hesitation. If a pencil is held between a book and the eye and double vision exists it does not interfere with the reading but if only one eye is competent reading is impossible as certain words will be cut out. This is an excellent test because the pencil can be thus used without attracting the attention of the malingerer. It must of course be held close to the eye and motionless. Lenses make still more difficult any fraud in connection with unilateral blindness. If, for example, a strong prism is placed in front of a sound eye, base up, and brought gradually in front of the eye from below upward double vision will occur before the base reaches the pupillary centre, one image directly transmitted, the other refracted. This being admitted by the patient, the prism is gradually pushed upward so as to obscure the pupil, producing a single image for that eye but if both eyes are competent a double image because of the different levels. If therefore a double image is still admitted as is almost invariably the case it proves vision in both eyes. Another test consists in using a 6 D convex glass upon the sound eye thus reducing the range of vision and making reading possible only at 17 cm. or less. The patient is asked to read and the book gradually removed when it will be found ordinarily that the range is greatly exceeded or variable. Still another test consists in introducing a red glass before one eye and a green one in front of the other; the patient is then asked to look at Snellen's colored test types. Only the colored letters that correspond to the color of the glass in front of the eye can be read by that eye and if one is blind unilaterally he will not even suspect the existence of letters other than those read by the supposedly sound eye. This test may also be made by using an ordinary pencil and a red pencil to make written characters of the two colors.*

Cancer.—By a proper irritation of an existing sore in certain regions such as the breast or lip or by attaching to the skin a section

*These tests are for the most part derived from Fuch's "Text Book of Ophthalmoscopy."

of the spleen a passable imitation of cancer may be attained but the success of such impostures seems incredible if any examination be made and the patient under proper observation.

Catalepsy.—This is imperfectly simulated as the maintenance of the forced postures characteristic of the true state is impossible, the arm or leg if extended soon tiring and dropping to the normal position.

Cerebral Concussion.—This condition is often feigned but it is only necessary to remember that the genuine lesion is associated with superficial respiration, pallor and moist skin and usually with nausea or vomiting.

Chorea is in itself difficult to simulate and readily detected if the subject can be observed when awakened from a sound sleep in which case he will for a moment forget his role.

Consumption.—Only the stethoscope can unmask the skilful malingerer who chooses this ailment and if as frequently happens there is actual bronchitis the exposure depends upon the absence of physical signs of infiltration or cavity formation and of fever or bacilli in contrast to the patient's apparent condition which is usually pitiable. The common method employed consists of:—feigned cough, bloody sputum, obtained by pricking the gums or when opportunity offers (as in hospitals) mixing ordinary sputum with blood or pus, and emaciation produced by abstinence from food, drinking quantities of vinegar or chewing and swallowing large quantities of tobacco.

Contractures.—Joint diseases with contracture or anchylosis offer an inviting field for the malingerer, but the joint in such cases is normal or becomes so if the patient is kept under observation and lacks implements and opportunity for producing fictitious lesions. Pain is frequently associated with the lesion and is often of such a nature as to at once expose the attempted deception. Muscular atrophy save that of disuse is lacking, the patient resists by evident muscular contraction attempts to move the joint and an anæsthetic reveals motility and exposes the fraud. One simple method is often effective with this class, viz., having the patient stand and then suddenly pushing him off his balance when the supposed crippled leg will usually be extended for support. Another method is to place the patient, standing on one leg, upon some high object, as a stepladder, the sound limb being slightly flexed; muscular fatigue will often force the patient to bring down the affected leg for support. Yet another consists in suspending a heavy weight over the contracted limb and it soon yields to the steady pull. Another old procedure involves the use of an Esmarch's bandage, which being

tightly applied, prevents the muscular action which maintains the false contracture.

Convulsions.—It is physically impossible to maintain for a long period the more violent convulsive movements and difficult to simulate correctly and in proper sequence those of the well known types. Nevertheless, even epilepsy may be exactly simulated by one having a thorough knowledge of the true disease and willing to go to the extent of biting the tongue and passing urine during the paroxysm.

Cutaneous Lesions.—These are readily simulated in many of their forms. A simple mustard plaster will produce erythema and may be cut in any form desired; pustules may be caused by croton oil; blisters by cantharides. Urticaria may be deliberately produced by eating certain substances known to produce them in the given individual (shell fish, strawberries, etc.). Pruritus offers no difficulties to the malingerer. Dermatitis and ulcer may be produced by acids or caustics; gangrene or the appearance of it by constricting bands and indeed nearly every skin disease including alopecia, bromidrosis and chromidrosis may be accurately feigned but are of little consequence. Detection of most of them is easy if a fixed plaster-of-Paris dressing can be applied, the patient being then unable to keep up the necessary irritation, and in the case of simulated gangrene, stripping the patient will disclose the cause and close confinement and observation will result in a disappearance of the lesion.

Diarrhœa and dysentery are both simulated, most commonly by mixing the fecal discharges with urine and adding blood from the finger or gums. Soap may also be employed and iron and bismuth taken by the mouth may add to the apparent abnormality. Such impostures ought never to be successful if a patient can be watched as the evacuations can be received in a pan and directly inspected.

Dropsy, Edema, Ascites.—Edema may be produced in an extremity by the use of a concealed ligature which however will leave its mark. It is even said that water has been injected directly into the peritoneal cavity.

Dyspepsia.—No disease is more easily simulated because of the predominance of subjective symptoms in the real disease. The introduction of a stomach tube after a test meal, if accompanied by a positive statement as to the certainty of its findings, may reveal the imposture, unless the simulator knows much of the vagaries of the disorders of digestion. Another successful method is the buttermilk cure with absolute rest in bed without means of diversion.

Dyspnœa on exertion can be clumsily simulated but with normal heart, blood, lungs, and kidney is disproved.

Calculi and gravel are frequently simulated, most commonly by urinary sediments, which are usually readily proven to be street gravel, brick dust, cinders or bone. In the female these substances are not infrequently actually introduced into the bladder or stored away in the vagina and used as occasion offers.

Epilepsy.—As before stated this disease can be but is with difficulty simulated. It is employed by professional thieves and beggars, as it draws a crowd, diverts attention and excites sympathy. The use of a pinch of snuff often suffices to expose the fraud as will the injection of apomorphia and occasionally the actual cautery or a threatened operation. Marked improvement under the administration of sodium chloride, the patient thinking that he is taking sodium bromide, may expose the supposed victim of chronic seizures.

The Eye.—Diseases of the eye, aside from the blindness already mentioned, are readily simulated, a great number of irritants being ready to hand. In these cases the rapid onset of inflammation and its prompt subsidence after the withdrawal of the irritant exposes the fraud. Chronic inflammation as induced by beggars may be extremely difficult to detect and is often carried to the point of ulceration of tissue.

Feigned Sleep.—It is said that in genuine sleep if the eye be suddenly opened even in the presence of a bright light there is transient primary dilatation of the pupil.

Fever.—Save in actual hysteria the simulation of fever is unsuccessful in the light afforded by clinical thermometers but nothing is commoner than the use of the hot water bag or a hot drink for the purpose of raising the temperature. Fever readings of any height are easily produced thus or by friction applied to the thermometer.* Chill is sometimes feigned but the malingerer is found hot and sweating under his blankets from the warmth and from his own exertions.

Fistula.—Anal fistula is readily and often successfully simulated by making a cut and treating it with some irritant such as carbolic or one of the strong mineral acids, but usually it is clumsily done and the imposture detected.

Fictitious Wounds.—Aside from mutilation under accident policies, fictitious wounds are not uncommon in connection with false

*In a recently observed private case a much desired winter trip to the South was obtained by this means.

charges of assault or in the case of those who seek to disguise their own theft by the pretense of attack and injury at the hands of burglars. They are also employed as a means of establishing self defense in cases of actual or attempted homicide. The most significant factor in their detection is in the comparatively trivial nature of the injury, the fact that knife wounds are inflicted in the region most accessible to the hand commonly used by the individual or as is often the case such cuts and gunshot holes are made in the clothing to intensify the appearance of a struggle, as should manifestly have involved the skin or body. Attempted suicide by throat cutting with the razor or knife may be denied or concealed but the characteristic incision obliquely downward and forward from the angle of the jaw on the side opposite the arm commonly in use is usually sufficiently characteristic.

Fractures.—Simulated fractures can only impose upon ignorance and are absolutely exposed by the X-Ray.

Headache.—Feigned headache can hardly be detected with certainty though close observation by the physician or nurse and the readiness with which sleep is produced may in some cases be important.

Heart.—Save in the rare instances of inhibition of the heart action, cardiac ailments are simulated only by producing palpitation and changes in the pulse rate. Sly compression of an axillary artery or the use of a concealed ligature may produce unilateral weak pulse. Digitalis in full doses may produce an apparent bradycardia, or, if pushed still further, an irregular and tumultuous heart action. Cardiac dilatation or hypertrophy, valvular murmurs and abnormally accentuated heart sounds are not readily simulated.

Hemorrhages.—The commonest are those simulating *hæmoptysis*, hæmatemesis, and hemorrhoids. The saliva may be clumsily covered with dyes, brick dust, etc., or, more commonly, mixed with genuine blood obtained by pricking the gums or fauces. If the symptoms of tuberculosis are absent both as to fever and physical signs a solution is relatively easy. *Hæmatemesis* may be simulated by pricking the gums and swallowing the blood which is afterwards vomited; it constitutes one of the most difficult of all impostures for the medical examiner, particularly if associated with feigned pain by one having a knowledge of gastric ailments. The sources of anal hemorrhage are readily detected by local examination, further they result from cutting or pricking the parts or from the introduction of blood, the lesion being evident upon removal of the blood which shows no possible source for

the hemorrhage. In some instances substances such as inflated fish bladder have been introduced to simulate hemorrhoids. They are readily detected by local examination.

Hernia.—Inflation of the cellular tissue of the scrotum and in some instances voluntary retraction of one testicle have been employed but the fraud is readily detected by the behavior of the supposed intestinal coil under manipulation, or the absence of one testicle.

Hydrocele.—Attempts to simulate this lesion by injecting water into the tissues of the scrotum are clumsy and unsuccessful and in any event readily detected by drawing off a portion of the fluid.

Hydrocephalus.—Pseudo-hydrocephalus has been produced by the injection of air into the cellular tissue of the scalp. The crackling on palpation and peculiar fascial distribution should at once excite attention.

Hydrophobia is frequently simulated but is usually greatly overdone and readily detected.

Hysteria may of course be readily simulated but is an absurdity.

Incontinence of Urine.—This is oftentimes extremely difficult to detect, but it is frequently possible to make the circumstances surrounding it so embarrassing as to expose the deception. The *steady drip* of overflow incontinence cannot be simulated if the patient is closely observed.

Insanity.—The following points may be of service in this frequently feigned disorder. *Melancholia.* A sudden onset with loss of memory and dementia at once proves the ailment spurious. *Mania.* A sudden onset lacking all relation between the surroundings and the patient's ideas, the relatively easily induced fatigue and natural sleep proves the falsity of the ailment. *Maniacal Frenzy.* Absence of a preliminary period of depression and loss of memory of events just prior to the attack are the two chief signs of fraud. *Paranoia.* The feigned disease is indicated by the obstrusiveness of the assumed delusions, the usual evidence of a capacity to weigh and judge the effect of statements made and the lack of elaborately systematized delusions. *Paretic Dementia.* A sudden onset, the absence of eye symptoms, the lack of true speech defects of the well known type are sufficient to establish the deception. *Loss of Memory.* The radical error of impostors is their failure to observe the fact that in all forms of dementia the loss of memory is of the senile type affecting recent events but not necessarily remote ones and often seeming to intensify those dating back to childhood and youth. In the less extreme form of genuine amnesia the

memory remains for striking events, the very ones in fact that a criminal seeking to escape punishment will forget.

Insanity is moreover extremely difficult to simulate and demands a knowledge of the various types, possessed by few. Not only is failure of memory usually exaggerated and often intermittent and inconsistent but the facial expression is difficult to assume and the demands upon the simulator for consistency in speech and action are seldom met. The person feigning dementia almost invariably pretends to have forgotten all events, but the true dement has usually a clear or partial recollection of prominent circumstances or events antedating his disease.

Jaundice cannot be successfully simulated because of the impossibility of maintaining the yellow discoloration of the conjunctiva when supplies are cut off. Turmeric has been used to color the skin, HCl added to the stools to make them clay colored, rhubarb ingested to give the urinary color and sometimes the eyes are deliberately inflamed to hide the deficiency in that region.

Joints.—See "contractures," "rheumatism" and "limping".

Limping.—Careful observation of a person using a cane or crutch may establish the fact that it is not being made a genuine support. Pretended stiffness of joints is revealed by the use of an anæsthetic and the absence of all inflammatory signs, joint crepitation, etc., will usually suffice to detect fraud.

Lumbago may be successfully simulated and is the favorite form, fortunately the treatment ordinarily pursued is sufficiently heroic to make it of short duration.

Ozena is said to have been feigned by the introduction of ripened highly odorous cheese into the nostrils.

Pain and Tenderness.—These are extremely difficult symptoms to detect the falsity of, being wholly subjective, yet if the patient be under close observation it is seldom difficult to expose the fraud. Few can carry on a conversation while the physician is examining the body and still be prompt to respond to pressure made with one hand over the supposed tender area while a more vigorous and obtrusive procedure is being carried on elsewhere with the other hand. Furthermore, the administration of morphia may suggest fraud, either by the relief of pain afforded by a small dose or a spurious one or by the assertion of persistence of pain or tenderness after the administration of full doses; nearly all cases of this kind are overdone and the pain is referred to vague, indeterminate, shifting or unusual locations. If, moreover, such a patient can be examined during sleep such as that produced by sul-

phonal or some other pure narcotic in moderate dose it will be found that the tender areas do not exist and that the pain which should prevent sleep does not affect it.

Paralysis.—This condition can hardly be feigned with success if the examination be thorough and in competent hands. Its description would involve a rehearsal of the signs and symptoms of the paralyses resulting from the lesions of individual nerves or nerve groups and the different areas of the brain and cord as well as the phenomena of hysteric paralysis. It may be well to remind the reader that in the last named disturbance loss of sensation in the extremities stops sharply at the junction of the limb with the body and if unilateral, is sharply limited by the median line and is likely to affect the whole body, including the face and scalp. Furthermore, the reflexes in this condition are likely to be bilaterally increased even though the paralysis is unilateral. Feigned anæsthesia is best detected by the unexpected application of the electric brush and it is impossible for a malingering patient to maintain the fixed boundary line of a true anæsthetic zone; repeated examinations revealing marked variations. So also partial general anæsthesia will almost invariably unmask feigned paralysis.

Peritonitis.—Is often simulated but seldom successfully; one of the most common lapses being a *restlessness* on the part of the patient sharply in contrast with the fixed position assumed in true peritonitis; the true facies and pulse cannot well be simulated.

Rheumatism can only be successfully feigned in its acute form by pounding the joints and this is seldom carried to the point of producing any misleading resemblance. The fever and peculiar sweating are beyond the patient's resource if he is under observation and the lesions rapidly fade.

Retention of Urine.—This cannot be simulated if the patient is carefully watched.

Sciatica.—This frequently feigned disorder is oftentimes readily detected by the clumsily described referred pain and points of tenderness. Few genuine cases lack maximum tenderness midway between the sciatic notch and the knee even though the other points are insensitive. In the genuine disease, moreover, if the extended leg is flexed upon the pelvis the pain is severe though the opposite movement causes little distress. In spurious cases the patient may walk upright though limping and feigning great pain and distress, whereas in the true sciatica the thigh is always more or less flexed and the body inclined forward.

Scoliosis.—Feigned scoliosis is almost invariably dorso-lumbar, the spinal axis is not changed as in the true ailment, the skin is more markedly involved and the secondary compensatory curve is absent.

Scurvy cannot be simulated except by deliberately taking mercury to the point of excessive salivation, and swelling of the gums will otherwise seldom or never be encountered.

Unconsciousness.—Aside from ovarian and supraorbital pressure in the hysterical cases, the application of a sharp electric current and similar drastic measures, by far the best procedure is that of administering ether when the patient will invariably struggle and probably talk during the stage of excitement unless the unconsciousness is real when there will be no response.

Venereal Diseases.—Such simulation is not infrequent in connection with blackmail. Artificial eruptions are as readily produced on the genitalia as elsewhere and a fair imitation of gonorrhœa can be produced by the use of caustics or other irritants whether in the female or in the male, but the absence of the gonococcus is readily established and the peculiar characteristics of a primary sore are not easily simulated.

Vertigo.—It may be impossible to detect spurious vertigo, though here as elsewhere close observation may result in catching the patient off guard.

Vomiting.—This may be a very difficult form of malingering to detect but is usually so causeless or so directly and immediately related to the taking of bland substances as to expose its real nature. It may often be detected by making the circumstances under which it would occur particularly embarrassing to the impostor. In a case of hysteric vomiting observed by the author a cure was effected by forcing the patient to go to the theatre for an evening.

Wry Neck.—This is usually readily detected by the muscular rigidity which follows any attempt on the part of the physician to correct the deformity. Furthermore the associated unilateral atrophy cannot be simulated and the condition disappears during sleep or anæsthesia.

Comment.—In conclusion one may say that in all simulated diseases detection depends upon (a) *the incongruity and lack of proper sequence in the symptoms presented;* (b) *the presence of a motive for simulation and* (c) *the patient's actions and the course of his ailment when under close observation and control or in the presence of shock, surprise, suggestion, and finally the use of drugs and anæsthetics.* By

far the most difficult cases are those occurring in the domestic circles where the physician must use great tact and judgment and is usually obliged to make the patient expose himself. It may be added that the ruse so often suggested of allowing patients to overhear conversations calculated to produce absolutely spurious symptoms is successful only in the case of stupid and ignorant patients, the brainy impostor being ordinarily prepared for such manœuvres. On the other hand in many instances the simplest of measures suffice for exposure if the patient is under control, among the commonest being the mere threat of operation, the administration of nauseating drugs or the establishment of an attenuated or unpleasant dietary and irksome restraint.

CONDITIONS SIMULATING DEATH.—Asphyxia.—In drowning especially, death is often only apparent and resuscitation possible after long periods of submersion (one hour or even more). The same is true of infants apparently born dead. Efforts to restore life in such cases should therefore be carried out for a long period before hope is abandoned.

Catalepsy.—In this condition the simulation of death is not ordinarily sufficient to lead to error although both respiration and circulation may be but faintly indicated, superficial reflexes lost, analgesia and anæsthesia present and the temperature decidedly low. In the "*trance state*" the simulation of death is much more perfect, the limbs being flaccid or showing a rigidity simulating rigor mortis, the face pale and the pupils often fixed in dilatation. In these cases the question of voluntary inhibition of the heart action and respiration is interesting and there can be little doubt that certain of the East Indian fakirs possess this power. In the case of Colonel Townsend as described by Cheyne, the mirror test and stethoscope failed to reveal any sign of respiration or heart action for fully half an hour.

Syncope.—(Fainting). As this represents a temporary failure of respiration and heart sounds and is associated with complete unconsciousness and pallor it perfectly simulates death but lasts ordinarily but a few seconds.

Signs of Life in Persons Apparently Dead.—This title seems to the author more appropriate than the usual one, viz.:—"Signs of Death." In the trance state or in those who can voluntarily inhibit heart action the pulse may be absent and the heart sounds inaudible, but the following signs will be present if the patient is living. 1*st. A deep red or purple color in the finger tips will be evident if a firm ligature be applied to the digit.* If applied to the wrist prominence of the

veins on the dorsum of the hand indicates life. The ligature must not be so tight as to completely cut off the circulation. *2nd. Several hours after a supposed death blood flows persistently from a cut.* A small artery should be chosen, not mere wet cupping or haphazard puncture. *3rd. If a needle thrust into the tissues and left for a time becomes oxidized, life is present. 4th. If any cloud repeatedly appears upon an ice cold mirror held close to the mouth there is respiration but its absence does not alone suffice to prove death. 5th. If a powerful vesicant produces redness or blisters there is life. 6th. If a body fails to take approximately the temperature of its environment 48 hours after cessation of rigor mortis there is life. 7th. Pupillary response to light shows life, its absence does not prove death. Several hours after death it is affected neither by atropin nor eserin. 8th. Persistence of the red in and visibility of the arteries of the optic disc* are signs of life as is *persistent clearness of the media* (6–8 hours after apparent death). *9th. A sensitive cornea is a sign of life, absence of the corneal reflex is not a sure sign of death. 10th. Presence of electric excitability in all muscles 24 hours after death indicates life.* (Usually lost in from 3 to 6 hours but retained for from 10–15 in certain cases.)

A BRIEF SUMMARY OF THE SYMPTOMS AND TREATMENT OF ACUTE POISONING.

ACIDS, MINERAL.—(HNO_3, H_2SO_4, HCl).

Symptoms.—Burning pain in the mouth, esophagus and stomach, vomiting of grumous liquid containing shreds of tissue; mind clear, intense thirst, dysphagia and usually dyspnœa or actual suffocation, scanty or suppressed urine, constipation and profound shock. *Acid Stains on Mucous Membranes:—Sulphuric.* White turning to dark brown or black. *Nitric.* White, then orange, then brownish red. *Hydrochloric.* White or gray. *Duration of fatal period:*—Several hours or *months* depending upon amount taken, etc.

Treatment.—Cardiac stimulants, morphia to relieve pain, baking powder or bicarbonate of soda in milk, egg albumin, carbonate of magnesia, calcined magnesia, mucilaginous liquids, sweet oil, olive oil, chalk, whiting, soap and water; *the stomach tube or pump being barred.* All treatment fails if pure acid has been swallowed in quantity.

ACONITE.—(*Aconitum Napellus*, monkshood, wolfsbane. Fatal dose: 80 min. of the tincture, maximum medicinal dose 1 to 5 min.).

Symptoms.—(Usually come on in a few minutes or may be delayed one hour or more.) Pallor, dryness and tingling of lips, tongue, pharynx,

then of extremities and finally of whole body, nausea, perhaps vomiting, pharyngeal constriction (subjective); slow, weak, later rapid and irregular pulse, subnormal temperature, slowed, shallow and irregular respirations, vertigo, dimness of vision, tinnitus aurium, clammy anæsthetic livid skin, dilated pupils if convulsions are present, profound prostration and death. *Fatal period* from 30 minutes to 5 hours.

Treatment.—*Absolute rest.* Evacuation of stomach by tube or stomach pump *if condition permits*, elevation of foot of bed, external heat, cardiac stimulants, artificial respiration, hypodermoclysis, hot rectal enemata. Tannin and charcoal may be given but are probably of little use. Large doses of digitalis seem effective (m xx–℥i).

ARSENIC.—(Fatal dose 1 to 2½ grs., retained).

Symptoms.—(After half an hour or more), thirst, epigastric pain and tenderness, nausea, vomiting, purging with tenesmus, muscle cramps, rapid labored respiration, incessant and violent retching, pulse weak and rapid, urine scant or suppressed, cold wet cyanotic skin, perhaps paralysis, convulsions and coma.

Variations—Cerebral symptoms may predominate, pain and vomiting may be absent or gastro-enteric symptoms be exceptionally violent. *The usual picture is that of cholera morbus. Average duration* in fatal cases between 8 and 72 hours, shortest 17 minutes.

Treatment.—Evacuation of stomach, bland and mucilaginous drinks, ferric hydroxide (ferric chloride precipitated by any alkali) in milk, barley water, etc., followed by castor oil.

ATROPIN.—(Belladonna. Fatal dose of atropin ½ gr.)

Symptoms.—(After ½–2 hours). Dry hot throat, dysphagia, vertigo, dilated pupils, blurred vision, flushed face, brilliant eyes, rapid pulse and respiration, active or even maniacal delirium, hallucinations, nausea, vomiting, scant, bloody or suppressed urine, sometimes strangury and priapism; scarlatiniform rash on face and neck, sometimes general; stupor and death. It should be remembered that 10–15 drops of the urine in such a case will dilate the pupil of any animal except birds and monkeys.

Treatment.—Evacuation of the stomach and bladder, morphine, pilocarpine, tannic acid, cardiac stimulants.

CANTHARIDES.—(Fatal dose 24 grs. or 1 oz. of tincture).

Symptoms.—Abdominal pain, vomiting, dysenteric purging, dysphagia, loin pain, dysuria, strangury and frequently priapism in the male, hot and swollen labia in the female or actual frenzied eroticism.

Treatment.—Evacuation of stomach, cathartics and bowel flushing, opium, cardiac stimulation, demulcent drinks but no oils, chloroform inhalations.

CARBOLIC ACID.—(Fatal dose usually ʒ–ʒi but less would probably suffice, depends largely on dilution and stage of digestion).

Symptoms.—Odor of acid on breath, whitened mucous membranes from contact. Burning pain throughout affected tract, vomiting, retching, contracted pupils, insensitive cornea, dyspnœa, respirations rapid and shallow, stertor, collapse, coma and perhaps convulsions. Urine scant or suppressed, smoky, sometimes black or bottle green. *Fatal period*—few minutes to several hours.

Treatment.—*Dilute alcohol*, whiskey, brandy in quantity, *then evacuation of stomach; epsom salts*, saccharated lime, soap and water may be used and demulcent drinks.

CAUSTIC ALKALIES.—(Sodium and potassium hydrate, ammonia).

Symptoms.—Same as corrosive acids except that there is dysenteric purging.

Treatment.—Oils, dilute vinegar, lemon juice, cardiac stimulants, *morphia*, milk, mucilaginous drinks. *Stomach tube contraindicated.*

CHLORAL HYDRATE.—("Knock out drops," etc.).

Symptoms.—Stupor or coma, pallor, either slow or rapid *feeble* pulse, slow respiration, pupils first contracted, later dilated, marked muscular relaxation, cardiac failure.

Treatment.—Cardiac stimulants followed by hot packs and brisk friction, artificial respiration, hypodermoclysis.

COCAINE.

Symptoms.—Nausea, vomiting, mental excitement, delirium, rapid pulse and respiration, dilated pupils, *fever*, convulsions, stupor, coma.

Treatment.—Cardiac stimulants, inhalations of chloroform to control convulsions, evacuation of stomach, morphia.

COLCHICUM.—(Fatal dose ½ oz. of wine or less).

Symptoms.—Those of a violent gastro-intestinal irritation with dilated pupils, cramps, perhaps delirium or convulsions, collapse, urinary suppression, partial or complete. *Fatal period* a few hours to several days.

Treatment.—Cardiac stimulants, hypodermoclysis, morphia.

CROTON OIL.—(Fatal dose 30 m.). Typical choleriform symptoms, collapse.

Treatment.—Symptomatic.

CASTOR OIL.—Same symptoms.

Treatment.—Symptomatic.

CORROSIVE SUBLIMATE.—(*Mercuric chloride.* Fatal dose:—3 grs.).

Symptoms.—Immediate choking, burning pain in epigastrium and esophagus, metallic taste, nausea, vomiting, persistent retching, dysenteric purging, distended tender abdomen, shock, dyspnœa, muscle cramps, syncope, stupor, convulsions. Salivation not immediate and sometimes absent. Poison has an acrid coppery taste as compared with arsenic and, in the case of the latter, symptoms are delayed and the stools are less often bloody.

Treatment.—Evacuate stomach, morphia, cardiac stimulants. Give white of egg in milk in quantity or flour paste.

FORMALDEHYDE.—This produces intense abdominal pain, *lachrymation and nasal irritation* (fumes), cyanosis and cardiac failure. The odor of the breath is characteristic.

Treatment.—Evacuation by stomach pump if apomorphia fails, ammonia by inhalation and the same drug (dilute) by the mouth if possible; morphine, cardiac stimulation

GELSEMIUM.—(Fatal dose ℥i of fluid extract or ℥iv of tincture).

Symptoms.—Extreme muscular weakness. *Oculo-motor paralyses* chiefly ptosis, *blurred vision*, fixed and dilated pupils, jaw drop, face pallid, congested, livid, cold sweat, impaired speech, dyspnœa (respiration slow), partial paralysis of extremities, marked cardiac depression, occasionally pharyngeal spasm or general convulsions. *Fatal period*, one to several hours.

Treatment.—Evacuation of stomach, *morphine freely*, cardiac stimulants, hypodermoclysis.

HYDROCYANIC ACID.—(Prussic acid. Fatal dose about ℥i of official preparation or 1 gr. of anhydrous acid).

Symptoms.—*Immediate, in large doses*, vertigo, muscular relaxation (patient falls), protruding eyes, locked jaws, slow, gasping respiration (respiration prolonged), dilated pupils, insensitive conjunctiva, pulse feeble, extremities cold, convulsions may occur, the face is livid and bloated, the lips foamy, the breath carries the distinctive odor of bitter almonds and death occurs in coma with *stertorous breathing* or convulsions.

Fatal Period.—*A few minutes.* Death is not instantaneous as is commonly believed.

Treatment is usually futile, impossible or unnecessary, the patient being dying, dead, or out of danger in a few moments.

LEAD ACETATE.—(Max. fatal dose about 1 oz.).

Symptoms.—Sweet metallic taste, violent vomiting and a particularly intense abdominal colic with a rigid retracted abdomen, intense thirst and obstinate constipation, or, more rarely, a diarrhœa with *black stools*, vertigo, convulsions and stupor or coma. The blue line may be present even in acute poisoning. *Fatal period*, a few hours to several days.

LOBELIA.—(Indian tobacco. Fatal dose ℥i of leaves).

Symptoms.—Nausea, vomiting, perhaps purging, cold sweats, contracted pupils, collapse, coma.

Treatment.—Evacuation of stomach. External heat, cardiac stimulants, morphia, tannic acid.

MUSHROOM POISONING.—(Usually *Agaricus muscarius or vernus*).

Symptoms.—Violent abdominal pain, vomiting, purging, mental and nervous excitement, dyspnœa, stertor, collapse.

Treatment.—*Large doses of atropia*,* hypodermoclysis, external heat, cardiac stimulants. Empty stomach if necessary.

OXALIC ACID.—(Frequently taken for epsom salts). *Fatal dose* ℥iv.

Symptoms.—(Immediate.) Violent vomiting, often bloody, or, acute pain in head, neck, back and limbs with tingling or numbness, tetanic spasms or convulsions, aphonia and marked cardiac weakness or collapse. Usually fatal within one hour, perhaps in a few minutes.

Treatment.—*Never give potassium or sodium salts* but rather chalk and magnesia in milk, oils, lime water, morphine and cardiac stimulants.

OPIUM.—(Fatal dose 4–5 grains; laudanum ℥i, or their equivalents. In children trifling doses have caused death. Habitues may use several hundred grains daily.)

Symptoms.—(Usually within from 3 to 30 minutes.) Somnolence, vertigo, stupor, coma, according to dose, the *pupils are contracted* ("pin point") and almost invariably *equal*, pulse slow and full, later weak and compressible. Respiration slow and stertorous, skin at first flushed

*This treatment was first successfully applied by Dr. Jacob E. Schadle, of St. Paul, who in a personal communication states that the dose should be gr. $\frac{1}{100}$ repeated as necessary and that gr. $\frac{1}{2}$ may be required during 24 hours.

and warm, later clammy, with diminishing respiratory rate. *Pupils may dilate* as death approaches. The fact that contracted pupils may occur in pontine hemorrhages and uræmic poisoning (Reese) must not be forgotten.

Duration of Fatal Period.—7–12 *hours.*

Treatment.—Evacuate stomach (apomorphia 1-10 gr., stomach pump, etc.). Hypodermic injections of atropine, administer strong coffee; exercise mild cases and dash cold water over chest to excite respiration. *Use artificial respiration* and cardiac stimulants. *Administer* 10 *grains of potassium permanganate in* 6–8 *ounces of water. Empty bladder, use hypodermoclysis.* In doubtful cases note odor of breath (laudanum) and search for hypodermic punctures, old or recent. Cocaine and adrenalin have been recommended and repeated lavage is valuable. Death occurs from respiratory failure indicated by increasing lividity and irregularity and slowness of respiration.

PHOSPHORUS.—(Fatal dose:—1-10 grain has killed).

Symptoms.—(Usually delayed several hours), "garlicky" breath, nausea, violent vomiting, abdominal pain, either diarrhœa or constipation, dilated pupils, cardiac depression. Vomited matter is green and is luminous in the dark; in protracted cases jaundice and hemorrhages from mucous membranes (especially hæmatemesis) or, under skin. Urine scant and albuminous, or suppressed.

Treatment.—*Ordinary oils must not be used.* Evacuate stomach by use of copper sulphate, purge freely and repeatedly; give *old* oil of turpentine in emulsion, albuminous and mucilaginous drinks (barley water, milk, flaxseed tea, egg albumin, etc.).*

POTASSIUM NITRATE.—(Nitre, saltpetre).

Symptoms.—Same as caustic alkalies but less marked, with tremor and perhaps convulsions and delirium.

Treatment.—*Evacuate stomach* and give milk, barley water, flaxseed tea, cardiac stimulants and morphia.

POTASSIUM CHLORATE.

Symptoms.—Vomiting, purging, æsthenia, delirium, spasm or convulsions, acute nephritis.

Treatment.—Same as preceding.

STRAMONIUM AND HYOSCYAMUS.—Same as belladonna.

STRYCHNINE.—(Nux vomica). Fatal dose gr. $\frac{1}{2}$.

Symptoms.—(After 15–30 minutes), *constriction of the throat or*

* H. C. Wood states that American oil of turpentime is valueless in this condition. Crude French oil is undoubtedly best.

subjective dyspnœa, muscular twitching of the face, followed by jerking of the head and extremities, and violent *tetanic* spasm (usually opisthotonos) associated with lividity. Contraction of the facial muscles produces the "risus sardonicus," there may or may not be locking of the jaw (if so *it comes on last and relaxes first in the spasms*), and as in hydrophobia, spasm may be induced by attempts to administer water. Relaxation follows in from ½ to five minutes and there is marked exhaustion and profuse perspiration. After a few minutes the spasm recurs and they increase in frequency and severity until death ensues from exhaustion or asphyxia. The special senses are exquisitely acute and a noise, draught of air, or sudden bright light may induce spasm; vomiting is usually absent. It should be noted as separating this condition from hysteria, that *the eyes are opened and the mind* clear until the last or until the supervention of asphyxia if the case thus terminates. *Unlike trismus* proper the *jaw muscles are involved late* and the convulsions are intermittent with periods of *complete* relaxation. *Fatal period*, 5 minutes to several hours.

Treatment.—Absolute quiet in a darkened room, evacuation of stomach *after chloroform inhalation has been started*, bromides and chloral, atropine.

TARTAR EMETIC.—(Fatal dose 1–2–40 grs.).

Symptoms.—Almost exactly as in arsenic poisoning with profound cardiac depression.

Treatment.—Same as arsenic save that tannic acid, not iron, is the antidote (strong green tea, etc.). *Morphia* and cardiac stimulants are indicated.

TARTARIC ACID.—(One oz. has proved fatal).

Symptoms.—Violent gastro-intestinal irritation.

Treatment.—Sodium bicarbonate, magnesia, chalk, best administered through stomach tube which permits emptying stomach of contents and generated gas.

TABLE OF APPROXIMATE METRIC EQUIVALENTS.

Grains or Minims	Grammes or (c.c.)	Grains or Minims	Grammes or (c.c.)
$\frac{1}{200}$	.0003	10	.6
$\frac{1}{100}$	.0006	12	.8
$\frac{1}{64}$	.001	15	1.0
$\frac{1}{32}$	.002	20	1.2
$\frac{1}{16}$	.004	30	2.0
$\frac{1}{12}$	.005	60	4.0
$\frac{1}{8}$	.008	120	8.0
$\frac{1}{6}$	.01	240	15.0
$\frac{1}{4}$	.015	480	30.0
$\frac{1}{3}$	.02	Ounces	
1	.06	2	60.0
2	.12	4	115.0
3	.2	6	170.0
4	.25	8	230.0
5	.3	10	280.0
6	.4	15	420.0
8	.5	20	568.0

CENTIGRADE AND FAHRENHEIT SCALES.

To convert Fahrenheit into Centigrade, subtract 32, multiply the remainder by 5, and divide the result by 9.

To convert Centigrade into Fahrenheit, multiply by 9, divide by 5, and add 32.

The following table shows the relation of degrees Fahrenheit to Centigrade

Centigrade	Fahrenheit	Centigrade	Fahrenheit
110	230	37	98.6
100	212	36.5	97.7
95	203	36	96.8
90	194	35.5	95.9
85	185	35	95.0
80	176	34	93.2
75	167	33	91.4
70	158	32	89.6
65	149	31	87.8
60	140	30	86
55	131	25	77
50	122	20	68
45	113	15	59
44	111.2	10	50
43	109.4	+ 5	41
42	107.6	0	32
41	105.8	− 5	23
40.5	104.9	−10	14
40	104.0	−15	+5
39.5	103.1	−20	−4
39	102.2	0.54° =	1°
38.5	101.3	1 =	1.8
38	100.4	2 =	3.6
37.5	99.5	3 =	5.4

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